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ESSENTIAL ORAL HYPERTHERMIA; REPORT OF A STUDY OF 25 CASES OF LOW GRADE 'FEVER' *

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THERE are few problems confronting the internist more challenging, vexing and yet intriguing than fever which persists for weeks or months without yielding a single clue as to its origin. Not infrequently, time or the laboratory establishes the diagnosis. With the elapse of a sufficient period of observation, some leading symptom or clinical finding becomes manifest or one of the usually numerous laboratory tests enlisted in the search yields the clue to solution.

The upper limit of normal oral temperature is almost universally regarded as 99° F. with rectal temperature one-half to three-quarters degree higher. Wright [1] accepts the range as 96.7 to 99° F. and 97.2 to 99.5° F. respectively, the rectal being generally considered the more reliable mode of determination. Kintner and Rowntree [2] believe that a continuous mouth temperature of 99° F. or over in an adult represents fever whether or not the person is nervous.

That elevation of oral temperature per se cannot be regarded as the sole criterion of either fever or organic disease may be adduced from the following survey of 25 cases admitted to the Medical Service of a general hospital with the diagnosis 'fever of undetermined origin.' Each had been observed at one or more hospitals for an average period of 16.3 weeks and had undergone extensive laboratory investigation for cryptogenic sepsis to no avail. Studies here disclosed that all exhibited an elevated oral temperature associated with normal rectal temperature. It is the purpose of this presentation to advance clinical evidence which favors an inherent vasomotor instability as the etiological basis of this paradox.

Consideration of these cases will be undertaken with regard to the following:

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1. Cause of original hospitalization
2. Symptomatology
3. Objective findings—a. Clinical
   b. Laboratory
4. Fever
5. Diagnosis
6. Management
7. Disposition and follow-up

1. *Cause of original hospitalization* (table 1). Twenty-two patients were originally hospitalized because of a well-defined febrile disease, and when all clinical signs of infection had subsided, it was noted that a persistent low-grade fever remained, resulting in a prolonged search for its etiology. Three patients were admitted primarily for treatment of disorders not usually associated with fever (duodenal ulcer, migraine, and lumbosacral strain), the elevated temperature being discovered upon routine check.

TABLE I

Cause of Original Hospitalization

Admission Diagnosis	Number
Acute upper respiratory infection	18
Bacillary dysentery	2
Malaria	1
Herpes zoster	1
Duodenal ulcer	1
Migraine	1
Lumbosacral strain	1
	25

2. *Symptomatology.* The usual symptoms of prolonged fever are malaise, exhaustion, anorexia, headache, nervousness, palpitation and weight loss. These complaints were registered by nearly all, particularly those who had been under observation for several months or more. Twenty-two experienced the sensation of fever to a varying degree; four of this group stated they had frequent chills. Such non-specific symptoms as fleeting precordial, muscle and joint pains, irritability, depression and insomnia were frequent. Hyperhidrosis of the palms and soles was a particular source of distress. Repeated interview, however, disclosed a constantly altering pattern of symptoms in the individual patient.

3. *Objective findings.* a. *Clinical:* Subjective complaints were rarely authenticated by objective evidence of disease. Despite the invariable complaints of anorexia and weight loss, comparison of weight upon admission to this hospital with that recorded at onset of illness revealed insignificant fluctuation. Fifteen were unchanged, five had gained and two had lost less than 5 per cent of their original body weight. They looked remarkably well despite protracted fever. Three patients were 12 to 15 pounds below their initially recorded weight. One of the latter had regained all lost weight during a 30-day furlough from another hospital, but gradually lost it again during 20 weeks of further hospitalization.

Dysorexia is a common sequel of prolonged hospitalization in the army and yet is frequently unattended by appreciable weight reduction. This is

due to curtailed activity with resultant diminished requirement for the high caloric intake customary during active field duty. The hypochondriac is inclined to view this physiologic loss of appetite with alarm.

The most constant finding was marked hyperhidrosis affecting the palms, soles and axillae. Although blood pressure showed little variation upon repeated recording, considerable fluctuation of pulse rate was in evidence. Tachycardia, however, did not always coincide with the period of maximum daily temperature, contrary to the pulse-temperature relationship characteristic of infectious fevers.

It is noteworthy that all patients whose hospitalization had been initiated by infection listed fever among their chief complaints. Of the three whose fever had been discovered in the routine temperature check, only one felt 'feverish' and he dated the onset of this symptom to his sixth week of hospitalization (case 25, table 2). Eight patients reported that they experienced a sudden sensation of 'burning up' at approximately the same time each day, usually in the late afternoon. This frequently but not invariably coincided with the peak of oral temperature, although rectal level was consistently normal.

Few of the numerous and varied subjective complaints could be corroborated by objective findings of deranged function or physical deterioration. This is not to be construed as evidence of malingering, but as the product of a pathological preoccupation with somatic sensations to which prolonged hospitalization contributes in no small measure. Thus two soldiers (cases 6 and 9, table 2) who were originally observed because of upper respiratory infection were eventually studied exhaustively with regard to diarrhea. Both had been aware of post-prandial evacuation of formed stools for many years and had displayed no apprehension regarding this frequency until after an inquiry had been begun regarding the cause of persistent fever, whereupon they developed marked 'bowel-consciousness.'

As a group they displayed lassitude, failed to engage in ward activities despite being ambulant, remained seclusive, asocial and exhibited considerable anxiety regarding the ultimate outcome of their disease. Three had purchased thermometers and were accustomed to take their oral temperature at frequent intervals during the day. Nearly all parried the suggestion that oral fever alone was not significant of disease with such reasonable rebuttals as, "Everybody knows a person with fever is sick" or "If I have fever I must be sick."

b. *Laboratory:* Although there are notable exceptions, it is anticipated that at some period during the course of a prolonged febrile disease progressive anemia, an alteration of the white blood count and Schilling index, or an increase of sedimentation rate would be found. Nevertheless in not a single instance were deviations from normal encountered upon repeated examination.

Prior to admission to this hospital, in addition to routine tests, studies included stools, sputum, gastric contents, liver function, prostatic smears,

TABLE II

Case No.	Age	Cause of Original Hospitalization	Period of Observation*	Temperature†		Final Diagnosis	Disposition	Subsequent Course	
				Oral	Rectal			Interval (months)	Report of Progress
1	21	Tonsillitis	36	99.8	99.4	N.C.A.‡	Discharge	4	Working on farm; fatigued and nervous.
2	24	Tonsillitis	8	99.4	99.6	Psychoneurosis (mixed)	Discharge	–	—
3	23	Tonsillitis	17	99.6	99.4	Psychoneurosis (mixed)	Duty	6	Oral temp. normal when released from hospital; doing army clerical work; fatigues; no symptoms of fever; has not taken temp.
4	22	Acute upper resp.	15	99.6	99.6	N.C.A.	Duty	3	No improvement; fatigue, headaches, and malaise; does not mention fever.
5	24	Acute upper resp.	4	99.6	99.4	No disease	Duty	–	—
6	20	Acute upper resp.	10	99.4	99.4	Psychoneurosis (anxiety)	Duty	4	Discharged from army after 2 months because of "nervousness."
7	26	Acute upper resp.	13	99.5	99.6	Psychoneurosis (anxiety)	Duty	3	Discharged from army because of "fatigue and nervousness."
8	23	Acute upper resp.	12	99.4	99.4	Psychoneurosis (anxiety)	Duty	–	Working as mechanic; feels well. Does not believe he has fever.
9	25	Acute upper resp.	20	99.8	99.5	N.C.A.	Discharge	5	Mouth temp. normal; fatigues and perspires easily.
10	26	Pharyngitis	18	99.6	99.6	Psychoneurosis (mixed)	Discharge	3	Tires readily; still nervous. No mention of temperature.
11	21	Acute upper resp.	14	99.6	99.4	Psychoneurosis (mixed)	Discharge	4	"Thinks his mouth temperature is normal." Improved, but tires easily. Working on farm.

* Weeks of continuous hospitalization prior to admission to general hospital.
† Average peak during 10-day period.
‡ Neurocirculatory asthenia.

TABLE II—*Continued*

Case No.	Age	Cause of Original Hospitalization	Period of Observation*	Temperature†		Final Diagnosis	Disposition	Subsequent Course	
				Oral	Rectal			Interval (months)	Report of Progress
12	20	Acute upper resp.	32	99.4	99.6	Psychoneurosis (anxiety)	Discharge	3	Managing grocery. Easily upset but feels stronger. No note about temperature.
13	21	Acute upper resp.	10	99.4	99.4	N.C.A.	Duty	–	—
14	22	Acute upper resp.	13	99.4	99.4	N.C.A.	Duty	–	—
15	26	Acute upper resp.	17	99.7	99.5	Psychoneurosis (anxiety)	Discharge	3	Improved but easily excited. Farming; no mention of fever.
16	24	Acute upper resp.	4	99.6	99.4	N.C.A.	Duty	–	—
17	28	Pharyngitis	5	99.4	99.6	No disease	Duty	5	Asymptomatic; oral temperature under 99°.
18	28	Acute upper resp.	8	99.4	99.5	N.C.A.	Duty	4	No improvement. Palpitation and fatigue.
19	23	Bacillary dys.	14	99.6	99.4	N.C.A.	Discharge	3	Easy fatigue and nervous under tension; farming; no note on fever.
20	28	Herpes zoster	23	99.8	99.2	Hysteria	Discharge	–	—
21	21	Bacillary dys.	50	99.3	99.4	Psychoneurosis (mixed)	Discharge	4	Still "nervous"; has no fever. Inspector in machine shop.
22	24	Malaria	40	99.7	99.4	N.C.A.	Duty	–	—
23	21	Duod. ulcer	10	99.4	99.4	Duod. ulcer N.C.A.	Discharge	4	Still has heartburn, palpitations, and fatigue; no mention of fever.
24	23	Lumbosacral strain	6	99.6	99.6	Psychoneurosis (mixed)	Discharge	–	—
25	27	Migraine	9	99.5	99.6	Hysteria	Discharge	–	—

basal metabolic rate, electrocardiogram, blood cultures, chest roentgenograms, and skin tests for tuberculosis, brucellosis, and occasionally coccidioidomycosis. Proctoscopy, bone marrow biopsy, spinal puncture and roentgen examination of the gall-bladder, gastrointestinal and urinary tracts were performed when indicated. All these diverse diagnostic procedures which were necessitated by the heterogeneous complaints that appeared to implicate various systems, were normal.

4. *Fever.* The transfer diagnosis, 'fever of undetermined origin,' was based upon the presence of a daily elevation of oral temperature above 99° F. without rectal controls. It was the probable, and not unreasonable, assumption of previous examiners that oral hyperthermia would be attended by a comparable rise in rectal temperature. The presence of an abnormally high oral temperature was confirmed by four-hourly determinations, but rectal temperature remained consistently normal. Temperature studies were conducted four-hourly over a 10-day period or longer, with close observation of the patient during the time of actual recording. Smoking, gum-chewing and physiologic post-prandial elevation of mouth temperature were ruled out as possible factors. Oral hyperthermia was considered established by the demonstration of repeated oral levels above 99.2° F. in the presence of rectal temperature of 99.6° or below. Cases exhibiting rectal temperature above 99.6° were excluded from this series, despite the presence of an even higher oral temperature and histories which were facsimiles of cases herein presented. Oral temperature rarely exceeded 99.8° F. but in three cases occasionally reached 100.2° F. Simultaneous recordings disclosed that at its daily peak, the oral was at the same level as or higher than the rectal by as much as 0.6 degree.

No constant relationship between mouth and rectal temperature could be established. Generally, oral was lower than rectal in the morning but equalled or exceeded the latter in the afternoon. Mouth temperature was usually between 97 and 98° F. at 7 a.m. and rose to its maximum peak at 3 p.m. Oral hyperthermia throughout the day was rarely exhibited for periods exceeding 48 hours. Complete remission of oral fever for one to two days was commonly noted but invariably followed by recurrence while under observation, with but one exception (case 5, table 2).

All patients had received one or more courses of the sulfonamides; a therapeutic test of emetine proved ineffectual in three. Tonsillectomy in three and removal of dental foci of infection were without benefit. Continuous bed-rest and mild sedation (phenobarbital) did not influence the course of 'fever.' Benzedrine sulfate was tolerated poorly by two subjects to whom it was administered to combat fatigue, and was discontinued after two days.

5. *Diagnosis.* Notwithstanding the oral hyperthermia, the maintenance of a normal rectal temperature is considered incompatible with the concept of fever on an infectious basis. The inability to confirm the presence of true fever, the normal laboratory studies, and the absence of significant physical

findings other than manifestations of autonomic lability strongly suggested that an anatomic basis for either the symptomatology or the pseudo-pyrexia was unlikely.

Many manifestations of neurosis, usually neurasthenic, or even psychosis may arise during the course of protracted fever, but these are, as a rule, transient and secondary to toxic insult. In patients of this series, however, a preponderance of neuropathic traits, and many of the symptoms for which they were being observed, had been present for many years prior to induction into the army. A history of vasomotor instability was obtained in 92 per cent evidenced by hyperhidrosis, palpitation on effort, easy fatigability, vertigo, and coldness of extremities, long antedating entry into military service. In 10 cases, these symptoms were sufficiently pronounced to warrant a primary diagnosis of neurocirculatory asthenia, typified by the following:

Case 13 (table 2). Pfc., aged 21, enuretic until 17 years, had always been underweight, 'nervous,' easily fatigued, subject to palpitation on slight exertion, and had noted perennial hyperhidrosis. He was inducted Oct. 15, 1943 and was admitted to a Station Hospital Nov. 2, 1943 complaining of sore throat, palpitation, cough and dyspnea. Physical examination was negative apart from mild tonsillitis, temperature 101.5° F. and pulse rate 125 p.m. Within 48 hours temperature had receded to 99°, but a daily rise to 99.8° was noted thereafter. He was transferred to a General Hospital Dec. 10, 1944 after various diagnostic procedures failed to reveal the cause of the fever. Blood counts, urinalyses, sedimentation index, basal metabolism, electrocardiogram, stools, spinal fluid, blood and throat cultures, non-protein nitrogen, sputum, gastric contents, prostatic smears, serological tests for syphilis, typhoid, tularemia, undulant fever and infectious mononucleosis; skin tests for tuberculosis, brucellosis and coccidioidomycosis, sternal marrow, allergy survey, chest and sinus roentgenograms, intravenous pyelogram and gastrointestinal series were normal. Glucose tolerance curve was somewhat flattened. Oral temperature continued to range between 98.2 and 99.8° F. Bed-rest, sulfathiazole, sedation, and finally tonsillectomy failed to influence the low-grade pyrexia. Following 250 days of hospitalization during which fever did not recede for periods beyond 36 hours, he was transferred to a general hospital with diagnosis 'fever of undetermined origin.' On admission patient appeared asthenic, weight 132 lbs. (height 67″). He stated that he had lost 'considerable' weight, but records proved his pre-induction weight to be 136 lbs. He complained of fever, chilly sensations, throbbing headache, palpitation, constant fatigue, anorexia, shooting pain in both groins, and precordial distress. Examination revealed nothing of note except for marked hyperhidrosis of palms, soles, and axillae with cold extremities, and considerable fluctuation of pulse rate during examination from 90 to 130 p.m. Rectal temperature during initial examination was 99.4° and oral 99.8° F. Four-hour determinations (simultaneous) for 10 days disclosed the following average daily recordings:

	Oral	*Rectal*
7 a.m.	97.6	98.0
11 a.m.	98.6	98.8
3 p.m.	99.4	99.4
7 p.m.	99.8	99.4
11 p.m.	99.4	99.2

On three occasions oral temperature, at its maximum daily peak, exceeded the rectal by 0.6 degree. The latter never rose beyond 99.6°. Cardiologist concurred in

the diagnosis of neurocirculatory asthenia, while psychiatrist found evidence of severe long-standing psychoneurosis, mixed type (neurasthenia and anxiety). Rectal temperature remained normal during his three-week stay at this hospital, but oral hyperthermia continued. As psychotherapy was not deemed beneficial in view of the chronicity of his symptoms, and in view of his meager military training (17 days), following repeated reassurance regarding the fever he was discharged from service. Four months later he reported that he was working on his father's farm and had no occasion to take his temperature or seek medical advice, but fatigue and nervousness persist.

Some cases were classified as psychoneurosis, mixed type, with anxiety the commonest component. It is emphasized that the longer the period of hospitalization, the more pronounced the evidence of neurosis. Indeed, the disparity between multitudinous complaints and meager clinical findings varied directly with the length of hospitalization. The preinduction history of this group is replete with neurotic trends and portends maladjustment to stress and strain of military life. Five were members of 'alerted' units when originally hospitalized while two had been evacuated from overseas, having been admitted to the hospital two weeks after arrival in the South Pacific. These are common factors bearing an important relationship to the progression of anxiety. Clear indication of long-standing autonomic dysfunction was present in all. Four soldiers exhibited conversion phenomena classically illustrated by the following:

Case 20 (table 2). Pvt., aged 28, a lawyer in civil life, had always been unaggressive, timid, self-conscious, dependent, and subject to phobias, palpitation, hyperhidrosis, vertigo, and easy fatigue since childhood. Despite one year of service with his Signal Corps unit he had received no promotions. Although he felt ill at ease due to necessity of obeying orders given by men he considered his intellectual inferiors, he stated that he shunned the responsibilities entailed by promotion. Two weeks after arriving in Australia he developed herpes zoster involving the right mid-abdomen and trunk and was admitted to a general hospital Nov. 1, 1943 with temperature 101° F. Temperature dropped to 100° in five days and rash disappeared in three weeks but his afternoon oral temperature rose daily to 99.8° F. Neurological examination was negative. Spinal puncture Nov. 22, 1943 revealed essentially normal fluid except for slight elevation of globulin (42 mg./100 c.c.). On the following day he complained of severe pain in his lumbar spine and weakness of his right leg. Within a week he developed an ascending weakness involving the right upper and lower extremities and right side of his face. A mask-like facies was noted. Deep reflexes were questionably diminished on the right, but all superficial reflexes were normal. A diagnosis of 'encephalitis' was at first entertained but later altered to 'Guillain-Barré syndrome.' Observation continued for four months, the loss of muscle function becoming gradually more marked, while daily fluctuation of oral temperature to 99.8° showed no recession. Because of failure to improve, he was evacuated as a litter patient to a general hospital April 4, 1944 with the diagnosis 'fever of undetermined origin, probably Guillain-Barré syndrome.' He was admitted by error to the General Medical section because of the primary diagnosis of obscure fever, and presented an apathetic picture, having lost 15 lbs. after five months in bed. Examination revealed mask-like facies, marked hyperhidrosis of hands and feet, right hemiparesis, normal reflexes, and a variable hypesthesia involving the entire right half of the body extending to the midline. The diagnosis of hysteria was considered most plausible in view of the normal reflexes and bizarre sensory disturbance. At initial examination oral temperature was 99.8° F.

and rectal 99.2° F. Four-hourly determinations proved that hyperthermia existed only orally. Neurologist confirmed the diagnosis of hysteria. Following appropriate suggestion, 7½ gr. sodium amytal were administered intravenously and patient rose from his bed, walked across the room and was able to swing his right arm for the first time in 10 weeks. On the following day he was able to feed himself and walk along the hall unaided. Restoration of full muscle strength was complete within one week and he was transferred to the Reconditioning Facility in the hope that he might eventually be returned to duty, having been assured an assignment consistent with his educational background. After three weeks, however, he stated that he 'couldn't make the grade' as he realized he was a complete failure in the army. Observations of both rectal and oral temperature following loss of conversion symptoms failed to indicate any change from their previous levels. Neurological examination was completely normal, and mask-like facies was no longer noted. Psychiatrist diagnosed 'conversion hysteria in an inadequate individual.' Lumbar puncture was not repeated to forestall the precipitation of further hysterical phenomena. He was discharged from the army and no further communication was received from him.

Case 3 (table 2). Pvt., aged 23, with eight months of service, developed sore throat and fever at a port of embarkation Dec. 22, 1943 and was admitted to a Station Hospital where 'acute tonsillitis' was diagnosed. Temperature was 103° F. on admission but receded to 99.2° F. in 72 hours. A low-grade irregular oral fever was noted thereafter, with daily rise to a maximum of 100° F. from a usually normal morning level. He was transferred to a General Hospital Jan. 15, 1944 for further investigation. Comprehensive laboratory studies failed to reveal an anatomic basis for the fever. Tonsillectomy and therapeutic trials of sulfathiazole and emetine were without benefit. Although his initial complaints were minimal, he developed an indefinite and ever-progressive pattern of symptoms including increasing fatigue, anorexia, headaches, chills and fever, pain in the middorsal spine radiating anteriorly and up his neck, precordial distress, and numbness of the right shoulder and arm. After 17 weeks of daily temperature elevation, he was transferred to a general hospital with the diagnosis 'fever of undetermined origin.' Examination on admission revealed excellent nutrition despite complaint of anorexia. Hypesthesia of the right arm to the elbow and of the right chest wall with sharp termination at the level of the elbow, not conforming to normal nerve distribution, was present. Moderate hyperhidrosis, tachycardia, and coarse tremor were noted. Simultaneous oral and rectal temperature determinations over a 10-day period revealed oral hyperthermia with normal rectal temperature. Patient revealed that he had always been restless, self-conscious, and tense. He had always been aware of palpitation and profound blushing when confronted with strangers. He had always been excused from physical training during his school career owing to 'rapid heart rate' and 'heart pain on exertion.' He had never been aware of fever until this period of hospitalization. Convalescence from 'colds' in civilian life had always been prolonged because of fatigue. Cardiologist at this hospital confirmed presence of a normal heart with neurocirculatory asthenia. Psychiatric diagnosis was 'psychoneurosis, anxiety and conversion.' Following repeated reassurance regarding the nature of his symptoms and oral fever, he was transferred to the Reconditioning Facility for six weeks, temperature recordings having been discontinued. Pain in the spine and numbness of arm and chest disappeared gradually without use of narco-synthesis. Convalescence was uneventful and he was returned to sedentary duties within the continental limits of United States. Studies of temperature were made for four days prior to release from this hospital and both rectal and oral temperatures were normal throughout the day. Communication from him after six months reported that he was doing clerical work at a fixed installation and that he had no occasion to seek medical advice since leaving the hospital although he felt tired throughout the day. He no longer experienced sensation of 'chills and fever' but had not rechecked either oral or rectal temperature.

It is considered that prolonged hospitalization is an important contributory factor in the progression of anxiety in many of this group. The constant stressing of temperature level, bed-side reference to fever of obscure origin, and numerous laboratory studies with inconclusive results instill the patient with the belief that he is suffering from a rare illness which has completely baffled his examiners. In suggestible individuals, this cannot fail to give rise to considerable apprehension and anxiety with the concomitant development of a wide variety of psychosomatic manifestations. This is illustrated by the following:

Case 17 (table 2). Pvt., aged 28, a radio school student with seven months of service, had always been active, aggressive, ambitious, and egocentric. He was admitted to a Station Hospital Dec. 10, 1943 because of sore throat and fever of 103° F. Temperature fell to 99.4° F. after 72 hours and he had no complaints but because of a daily rise of oral temperature he was retained in the hospital despite his protests. Following one month of investigation which failed to disclose a cause for fever he was returned to duty with instructions to report to the dispensary for daily temperature observation at 4 p.m. Because the latter was noted to be 99.8° on several occasions he was readmitted to the hospital for three weeks where further studies failed to uncover an infective focus. As he had no symptoms, he was again returned to duty. Owing to the inconvenience entailed by daily visits to the dispensary, he purchased a thermometer and recorded his temperature several times daily. About Feb. 15, 1944 he began to experience 'chills and fever' at approximately 6 p.m. each day, at which time his oral temperature reached a peak of 99.6° F. Because of this, he retired at 5:30 p.m. nightly to 'await his chill,' which would invariably leave him completely fatigued. On March 1, 1944 he requested readmission to the hospital because of increasing fatigue, fever, headache, anorexia and insomnia. After a protracted investigation, he was transferred to a general hospital April 7, 1944 with the diagnosis 'fever of undetermined origin.' Physical examination was entirely negative. Patient was observed taking his own temperature frequently during the day and this practice was ordered discontinued. Our own studies over a 14-day period confirmed the presence of oral hyperthermia with consistently normal rectal temperature. Afebrile periods up to 36 hours were noted on three occasions. It required considerable reassurance to convince him that he had neither an infectious fever nor underlying organic disease. This was accomplished in part by permitting him to take his own rectal temperature recordings. His daily 'chill' occurred even during afebrile periods. Amphetamine sulfate, administered in 10 mg. dose on two occasions, produced tremor, palpitation, tachycardia (102 p.m.) and profuse perspiration with an increase in oral temperature from 98.4° and 98.6° respectively, to 99.2° F. in one hour. A definite history of autonomic instability or neurotic traits prior to his present illness could not be obtained. Following three weeks of graduated exercise on the Reconditioning Facility, during which temperature studies were discontinued, all symptoms except fatigue disappeared. Maximal mouth temperature was 99.2° with 99.4° by rectum during a four-day period of observation, after the above regime. He was returned to duty with the diagnosis 'observation, no disease found.' Five months later he reported that he was asymptomatic and that his mouth temperature had been repeatedly under 99°.

Thus, it is possible that a transient tension state with attending hypochondriasis had been created in this self-centered soldier by the repeated suggestion of fever due to an obscure smoldering disease, although the oral hyperthermia was possibly the result of autonomic instability following a febrile disease (tonsillitis).

6. *Management.* Four-hourly temperature determinations for 10 days were considered adequate to rule out the possibility of true pyrexia since none had been afebrile longer than two days prior to admission to this hospital. Thereafter only rectal recordings were made twice daily to impress upon them that they were not 'fever problems,' as these patients are prone to be 'thermometer-conscious.' When an anatomic basis for their symptoms had been clearly eliminated, detailed psychiatric and cardiovascular studies were instituted, patients remaining on the general medical section until final evaluation had been formulated. Those transferred to the Reconditioning Facility were recalled to the Medical Service for a final period of observation preparatory to return to duty or separation from service if they failed to qualify for even sedentary duty.

7. *Disposition and follow-up* (table 2). During the period covered by this survey, group psychotherapy had not yet been given general application in the reconditioning program, and it is possible that this form of therapy might have yielded a higher salvage rate. Furthermore, the majority of those soldiers returned to duty gave evidence of poor adjustment to military routine, portending future difficulties and hospitalizations.

Reports were received in three to six months from nine of those discharged from the army. All were engaged in civilian occupations and had required no subsequent medical care. Three state that their oral temperature is normal; six omit mention of the latter. All comment vaguely regarding 'nervousness.'

Ten were returned to 'limited service' despite persistence of many of their symptoms present on admission. Oral temperature had reverted to normal in but one (case 3). Two were discharged from the army within two months, both soldiers reporting that they had been reassigned to duties which were too strenuous. Four others were traced three to six months after release from this hospital. Two stated they felt well and did not believe they had fever, while two reported they were 'no better.' Four of this group have not been heard from.

Communication was received from one of the two men returned to full duty (case 17) with a wholly favorable course.

No definite conclusions are warranted from these meager returns, and it is believed that the majority of those who reported the absence of oral fever were guided subjectively rather than by thermometer readings. In general, it appears that those patients who had secured release from service had fewer symptoms than those returned to duty, while none of either group, with whom contact had been established, developed progression of symptoms or physical deterioration.

Discussion

Fever is almost invariably regarded as indicative of organic disease. Wechsler [3] categorically states that fever is never hysterical. Nevertheless Eichelberg,[4] Potosky,[5] Deutsch [6] and others have advanced convincing proof

that fever may have a hysterical origin and recede under hypnosis. In their case reports, however, elevation of rectal temperature was always noted. The cases herein described never presented a rectal temperature above normal while under observation at this hospital. In view of the fact that the height of the oral temperature persisted at the same level as at previous hospitals, it is reasonable to assume that a normal rectal reading had been present for some period prior to admission here.

In explaining the cause of this temperature paradox it must be assumed that in addition to an increase in oral circulation there must be a variation from the normal distribution of blood in the oral and rectal tissues.

That irradiation of impulses from higher centers may produce profound effects on peripheral circulation is evidenced by such phenomena as syncope, pallor, blushing, and erection. Thus, psychic stimuli exert an influence upon the vascular mechanisms and reciprocal relations which exist between such vascular beds as the muscular and cutaneous tissues, the splanchnics and skin.

Following a febrile disease, the heat regulating mechanism is notoriously unstable and reacts sharply to both emotional and physical stimuli. Thus in psycholabile individuals, temperature rise following infection is not necessarily the result of psychic injury but may reflect the increased lability of the heat-regulatory mechanism acquired through organic disease.

It is conceivable that following an initiating febrile episode in an individual with autonomic instability, the heat regulating apparatus acquires a lability which may persist for long periods as a result of repeated or continuous situational and environmental stress. In some subjects, such psychic stimuli may produce an alteration of the usual reciprocal relation between vascular beds resulting in increase of oral circulation through selective sympathetic stimulation. The imposition of a strong element of suggestion will not alone prolong this response but, in a suggestible subject, may result in actual somatic sensation of 'fever.'

However, although it would appear that an infection resulting in authentic pyrexia, even though of short duration, initiated this response in 88 per cent of this series, the possibility must be entertained that oral hyperthermia antedated the infection which resulted in hospitalization. This is further suggested by the accidental discovery of 'oral fever' of a similar type in the three cases in this series who were being observed for non-infectious disorders, but who displayed considerable psycholability. It would bear out the observations of Friedman [7] who noted episodic oral hyperthermia in 36 per cent of 30 cases of neurocirculatory asthenia without known antecedent infection. In four cases there was neither subsidence nor progression during an observation period of 110 days. Correlating rectal temperatures were not reported in his study, but the cases surveyed appear to be facsimiles of those herein presented.

Nevertheless, that an antecedent stimulus, usually a febrile episode of infectious origin is an important factor in the elaboration of this syndrome is suggested by the absence of thermal abnormalities in the patients observed

on the neuro-psychiatric wards at this hospital, and in whom autonomic instability is frequently associated with anxiety. Cases of oral hyperthermia have been discovered among patients referred to this department from other sections of the hospital for evaluation of persistent low-grade oral fever. These oral hyperthermics invariably emanated from the contagion and surgical wards, and in the latter instance, temperature elevation appeared to have originated post-operatively.

Finally, it must be considered that a small percentage of individuals may normally exhibit oral hyperthermia without relation to infection or to vasomotor or psychic instability.

Comment

In large medical installations where temperature is estimated almost exclusively per os, due to the facility of this method, elevations, unless attended by unequivocal signs of infection should be controlled by rectal recordings. Where the latter remain consistently normal, the patient may be considered afebrile. This does not imply that associated symptoms be disregarded, but that careful clinical consideration be given before instituting a prolonged hospital regime and a medley of diagnostic procedures. The associated psychic stress may so harass a patient as to prolong hyperthermia and produce symptoms through pain and fear. In the management of patients with this syndrome the best weapon in allaying apprehension and engendering confidence is an attitude of the clinician indicating complete familiarity with its manifestations. The significance of elevated mouth temperature should be minimized while stressing the normal rectal level as being incompatible with fever on an organic basis. The practice of taking frequent oral temperature recordings should be discouraged when this paradox has been uncovered.

The term 'fever' is not ordinarily applied to the hyperthermia in the tissues involved by localized inflammation such as a furuncle or phlebitis. It is thus suggested that the syndrome herein presented of oral fever with normal rectal temperature be designated as 'essential oral hyperthermia.' The designation 'fever of undetermined origin' should be relegated to such cases which display elevation of temperature of the entire body rather than selective areas. While clinical impressions thus far strongly indicate that in the overwhelming majority of cases it stems from an inherent vasomotor instability, study of a larger series with adequate controls over a long period is desirable before it can be conclusively established to be solely on a functional basis.

Summary and Conclusions

1. Oral temperature may be elevated to levels generally regarded as abnormal, and exceed coexisting rectal temperature for prolonged periods without demonstrable organic disease. A series of 25 cases exhibiting this thermal paradox is presented.

2. Although the pathogenesis of this condition is not clearly established the weight of clinical evidence indicates that in the large majority of cases this response is initiated by an antecedent infection in psycholabile individuals which renders the heat regulating mechanism unstable. A reversal of the usual reciprocal mechanism existing between vascular beds by sympathetic stimulation from higher centers may produce increase of oral circulation.

3. Prolongation of this response and the elaboration of somatic sensation of fever may result from apprehension engendered by prolonged hospitalization, and the repeated suggestion to the patient of an underlying obscure disease.

4. The possibility of oral hyperthermia preëxisting the infection, in subjects with vasomotor instability or even being exhibited by normals is considered.

5. The demonstration of elevated oral temperature without associated evidence of infection should be invariably controlled by rectal temperature determinations. If the latter remain consistently normal, prolonged hospitalization and laboratory search for cryptogenic sepsis is unwarranted.

6. The term 'fever of undetermined origin' is misleading and is not applicable to localized temperature elevations.

7. "Essential oral hyperthermia" is suggested as a more accurate designation of this benign thermal disturbance.

8. An arbitrary level of 99° F. as the maximum limit of normal oral temperature is not valid.

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THE NEPHROTIC PHASE: ITS FREQUENCY OF OCCURRENCE AND ITS DIFFERENTIAL DIAGNOSTIC VALUE IN DETERMINING THE NATURE OF THE RENAL LESION IN 120 PATIENTS WHO DIED OF RENAL FAILURE*

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INTRODUCTION

THE term "nephrotic phase" is used to describe a syndrome occurring during the course of chronic glomerulonephritis and characterized by albuminuria, hypoproteinemia, hypercholesterolemia and edema. In general, these features have served as the criteria for diagnosis.[1, 2] The degrees of hypoproteinemia, proteinuria, edema and lipemia necessary for the diagnosis have not been clearly defined.

There is agreement over the occurrence of the nephrotic phase in particular types of renal diseases which terminate in renal failure. Baehr [3] noted that "every case of glomerulonephritis has a nephrotic element." Christian [4] agrees with this opinion. The rarity of the nephrotic phase in arteriolarnephrosclerosis is noted by Loeb [5] who states that, "The nephrotic syndrome with marked depression of the albumin-globulin ratio also serves as a distinguishing feature since it does not occur in arteriolarnephrosclerosis." Weiss and Parker [6] and Fishberg [7] mention the infrequency of generalized edema during the course of pyelonephritis in the absence of cardiac failure. Recently, Mansfield, Mallory and Ellis [16] have again called attention to the absence of the nephrotic phase in a series of patients with arteriolarnephrosclerosis and pyelonephritis. It seems clear from the opinions expressed above that the nephrotic phase is a common feature of chronic glomerulonephritis and, if it occurs at all, a rare phenomenon in arteriolarnephrosclerosis and chronic pyelonephritis. However, the factual data to support this opinion are scanty in the medical literature.

The present study, therefore, was undertaken to determine the frequency of the nephrotic phase in a large series of patients dying of renal failure caused by chronic glomerulonephritis, arteriolarnephrosclerosis and chronic pyelonephritis. The differential diagnostic value of this syndrome would then be apparent if it were found to occur exclusively in the course of only one of these entities.

DATA

The case histories of 120 patients dying of renal failure were reviewed. These consisted of 50 instances of chronic glomerulonephritis, 50 of arterio-

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From the Research Service, First (Columbia) Division, Goldwater Memorial Hospital, Department of Hospitals, and the College of Physicians and Surgeons, Columbia University, New York City.

larnephrosclerosis and 20 of chronic pyelonephritis. Autopsy was conducted in each instance. One hundred and six patients had been observed at the Presbyterian Hospital and 14 patients had been observed at the First (Columbia) Research Division of the Goldwater Memorial Hospital. The diagnosis of renal failure prior to death was made on the basis of the clinical picture and diminished or diminishing renal function as determined by one or more of the usual tests. Although the degree of renal failure in these patients varied over a wide range during the period of observation, no patient was accepted for this study unless the evidence of renal insufficiency was obvious.

The postmortem examinations were done by members of the Department of Pathology of the College of Physicians and Surgeons of Columbia University. The interpretation of the pathologist is accepted as the final diagnosis in each case, although there were differences of opinion between the clinician and pathologist in a few instances. When the 120 consecutive case histories were found to conform to (a) the pathological diagnosis of either chronic glomerulonephritis, arteriolarnephrosclerosis or chronic pyelonephritis, and (b) the presence of a period of renal insufficiency prior to death, a study of each chart was made to determine any evidence which would indicate the presence of the nephrotic phase during the period of clinical observation. This evidence is now presented in reference to each finding.

Those patients were considered in a nephrotic phase who showed albuminuria, hypoproteinemia, hypercholesterolemia and edema.

Serum Proteins

A comparison of the serum protein values in the three groups of patients is shown in table 1. In those patients with chronic glomerulonephritis there were frequent determinations made throughout the period of observation.

Table I

Serum Protein Values During the Clinical Course of 120 Patients Dying of Renal Failure

	Chronic Glomerulonephritis	Arteriolar-nephrosclerosis	Chronic Pyelonephritis
Total number of patients	50	50	20
Number of patients with serum protein determinations	45	42	11
Number of patients with normal serum protein levels	9	25	5
Number of patients with serum proteins below 6 gm. %	36	17	6
Number of patients with serum proteins below 5 gm. %	22	2	3
Number of patients with serum proteins below 6 gm. % in absence of cardiac failure or malnutrition	26*	1	1

* Two of these determinations occurred in patients not having nephrotic phase. The total serum protein level was 5.9 per cent in one patient and 5.3 per cent in the other.

In the other two groups the number of determinations per patient was comparatively small. This was due to the fact that a decrease in the serum protein level in the latter two groups was seldom expected. The table indicates that roughly four-fifths of the patients with chronic glomerulonephritis had a decreased serum protein value whereas about two-fifths of those with arteriolarnephrosclerosis or pyelonephritis showed a value below normal. The degree of alteration was much greater in those patients having chronic glomerulonephritis.

These data corroborate the findings of many others. It has been previously noted by Peters and Van Slyke,[8] and Fishberg[7] that hypoproteinemia is a frequent finding at some time during the course of chronic glomerulonephritis. On the other hand Linder, Lundsgaard and Van Slyke,[9] Peters,[10] Van Slyke, Stillman, Möller and his associates[11] have shown that patients with arteriolarnephrosclerosis have normal serum proteins if nutrition is adequate and cardiac failure is absent.

Serum Cholesterol

A comparison of the serum cholesterol values in the three groups of patients is shown in table 2.

Table II

Serum Cholesterol Levels During the Clinical Course of 120 Patients Dying of Renal Failure

	Chronic Glomerulonephritis	Arteriolar-nephrosclerosis	Chronic Pyelonephritis
Total number of patients	50	50	20
Number of patients with cholesterol determinations	36	15	9
Number of patients with cholesterol over 300 mg. %	26	3	1
Number of patients with cholesterol below 300 mg. %	10	12	8

Although the determinations of serum cholesterol were limited in number, the data in the table suggest that hypercholesterolemia is a far more frequent finding in patients having chronic glomerulonephritis than in the other two groups of patients. These data are consistent with the opinion expressed by other workers. Peters and Van Slyke[8] state that serum lipids as well as serum cholesterol are elevated in nephrotic types of glomerulonephritis and less commonly in non-nephrotic types. Steiner and Domanski[15] found that serum cholesterol levels exceeded 300 mg. per cent in 30 of 54 patients having chronic glomerulonephritis. Bloor,[12] Denis[13] and Page, Kirk and Van Slyke[14] have shown values within normal limits in patients with arteriolarnephrosclerosis.

Proteinuria

The incidence of severe proteinuria occurring during the course of clinical observation in the three groups of patients is shown in table 3. Differentiation is made between proteinuria due to renal disease alone and pro-

TABLE III

Incidence of Marked Proteinuria During the Clinical Course of 120 Patients Dying of Renal Failure

	No. of Patients	4+ Proteinuria*	Heart Failure at Time of 4+ Proteinuria
Chronic glomerulonephritis	50	40	10
Nephrotic phase	27†	25	4
Non-nephrotic phase	23	15	6
Arteriolarnephrosclerosis	50	16	15
Chronic pyelonephritis	20	3	1

* This refers to a period during the patients' observation when consecutive findings of 4+ proteinuria were present.

† 24 patients who had nephrotic phase during the period of observation; 3 patients who had a history of nephrotic phase before the period of observation.

teinuria observed when complicated by heart failure. It is seen from this table that: (1) marked proteinuria is more frequently found in chronic glomerulonephritis than in arteriolarnephrosclerosis or chronic pyelonephritis, (2) when the proteinuria of the nephrotic phase occurred, it was usually unassociated with cardiac failure during specified periods of observation, (3) when severe proteinuria occurred in arteriolarnephrosclerosis there generally was associated cardiac failure. The urine proteins were determined by the heat and acetic acid test. Peters and Van Slyke [8] have observed that, "Among patients with chronic nephritis those with degenerative nephritis or the chronic active stage of hemorrhagic nephritis excrete the largest amounts of protein. . . . In the nephrosclerotic type of disease, the quantity of protein in the urine is usually small, sometimes almost undemonstrable." Recently Mansfield, Mallory and Ellis [16] have made similar observations.

EDEMA

The incidence of the history of edema occurring in the three groups of patients is summarized in table 4. It may be seen from the table that: (1) in

TABLE IV

Presence of Edema During the Clinical Course of 120 Patients Dying of Renal Failure

	Total	Edema	Anasarca	At Time of Edema	
				Heart Failure	Malnutrition
Chronic glomerulonephritis	50	37	13	10	7
Nephrotic phase	27*	26	12	4	2
Non-nephrotic phase	23	11	1†	6	5
Arteriolarnephrosclerosis	50	27	2†	21	6
Chronic pyelonephritis	20	12‡	0	6	4

* 24 patients observed with nephrotic phase as defined in this paper. 3 patients had substantiated histories of nephrotic phase.

† Heart failure cause of anasarca.

‡ One patient had cirrhosis of the liver and low serum proteins. One patient had edema from forced saline therapy.

patients having the nephrotic phase of chronic glomerulonephritis, edema is frequently observed in the absence of cardiac failure or malnutrition, (2) when edema is present in arteriolarnephrosclerosis or chronic pyelonephritis, heart failure or malnutrition is frequently present, (3) anasarca occurred chiefly in the patients having the nephrotic phase. Twelve of 27 patients having the nephrotic phase of chronic glomerulonephritis had anasarca, the remaining 15 patients having only moderate or slight edema. In this latter group the serum protein level, the hypercholesterolemia, and marked albuminuria favored the diagnosis of the nephrotic phase.

TABLE V

Incidence of the Nephrotic Phase during the Clinical Course of 120 Patients Dying of Renal Failure

	Total Cases	With Nephrotic Phase	% With Nephrotic Phase
Chronic glomerulonephritis	50	27	54
Arteriolarnephrosclerosis	50	? 1	? 2
Chronic pyelonephritis	20	? 1	? 5

DISCUSSION

It is shown in table 5 that 54 per cent of the patients with chronic glomerulonephritis had a typical nephrotic phase. Forty-eight per cent exhibited the quadrad of edema, hypoproteinemia, marked proteinuria and hypercholesterolemia during the period of clinical observation. Six per cent had a history of the nephrotic phase in the past without a history of protein starvation or cardiac failure. In another 20 per cent, one or more of the classical signs were seen; namely, edema, hypoproteinemia, cholesterolemia or heavy proteinuria or combinations of these changes. These alterations could not be explained on the basis of malnutrition, cardiac failure or another disease process producing an increase in the serum cholesterol level. These findings, with a few exceptions, could not be duplicated in the group of patients with renal failure due to nephrosclerosis or pyelonephritis. In only 26 per cent of the patients with chronic glomerulonephritis was there a complete absence of the findings usually associated with the diagnosis of the nephrotic phase. The period of clinical observation in these cases was shorter than that in the nephrotic phase group. In 21 of the 27 patients having the nephrotic phase, the period of observation was for one or more years. However, of the 23 patients not having the nephrotic phase, there were only six patients with a follow-up period of one or more years. Thus there is the possibility of the nephrotic phase having occurred in certain of the latter patients at some time before the period of observation.

In a parallel study, it was found that of 11 patients with chronic glomerulonephritis who were observed for a long period at the Columbia Research Service of the Goldwater Memorial Hospital, 10 exhibited classical findings of the nephrotic phase. The remaining patient, who was shown to

have intercapillary glomerulonephritis at autopsy, was said to have had a mild nephrotic phase prior to admission. However, he manifested no edema during a one-year period with us and his serum albumin value was never below 3.8 gm. per 100 c.c. The degree of proteinuria was four plus. The serum cholesterol level varied between 377 and 610 mg. per cent, with a mean of 479 mg. per cent. The basal metabolism was normal and there was no evidence of diabetes mellitus.

During this study the question of a repetitive nephrotic phase was raised. In the present series of case histories no evidence was found of the nephrotic phase occurring more than once during the course of glomerulonephritis. The duration of the nephrotic phase ranged from two months to five years. The average duration was 22.5 months.

In the series of 50 patients who died of arteriolarnephrosclerosis, there is one patient having the nephrotic phase (table 5). Although there is no doubt as to the presence of the nephrotic phase, there is doubt as to the diagnosis of the renal disease in this patient. Description of the pathological changes in this case history indicated that the findings were of a borderline character which are difficult to classify. There is only one patient in the pyelonephritis series with findings suggestive of the nephrotic phase. On closer analysis these findings are not exactly in keeping with our definition, for the marked proteinuria was not present at the same time as the hypoproteinemia and hypercholesterolemia. This study thus confirms the opinion that the nephrotic phase is a frequent feature of chronic glomerulonephritis and a rare phenomenon in arteriolarnephrosclerosis and chronic pyelonephritis. The differential diagnostic value of the nephrotic phase is thus apparent, for once established, it helps to identify the renal lesion as that of chronic glomerulonephritis.

Conclusions

1. The nephrotic phase is defined as the concomitant occurrence of edema, hypoproteinemia, hypercholesterolemia and a marked proteinuria. Difficulty is encountered in establishing this diagnosis in the presence of cardiac failure and severe malnutrition.

2. The opinion is confirmed that the nephrotic phase is a frequent finding during the course of chronic glomerulonephritis and a rare phenomenon during the course of chronic pyelonephritis or arteriolarnephrosclerosis. The presence of the nephrotic phase is therefore a useful differential diagnostic factor in the diagnosis of the cause of renal failure.

3. The nephrotic phase may extend over a number of years. The average duration in these patients was 22.5 months.

4. No evidence of a repetitive nephrotic phase was observed in this series of patients.

We are indebted to members of the Department of Pathology of the College of Physicians and Surgeons for the use of their records.

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MEDICOLEGAL PROBLEMS IN DISTINGUISHING ACCIDENT FROM SUICIDE *

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I

INTRODUCTORY †

SUBSTANTIAL rights, duties, and penalties created by statute, contract or the common law and affecting life, liberty, and property often depend upon whether a death was caused or hastened by disease, accident, suicide or homicide or some combination of these causes. In the investigation of any sudden death one should consider all possibilities, and never, at the outset, arbitrarily limit the scope of the inquiry.[1] Appearances mislead and deceive quite as often in medicolegal investigations as in clinical medicine, and in the former there are additional complicating factors: purposeful simulation to hide crime or obtain money, and the fact that ultimately the real cause of death may be determined by a jury, administrative tribunal or court of law.

With these preliminary admonitions in mind, it is usually, but not always, possible to limit an inquiry in any one case to one of these four groups: (1) disease or accident, (2) accident or suicide, (3) accident or homicide, and (4) suicide or homicide. This paper is confined to the second class of cases.

Suicide is voluntary, willful self-destruction, the act of designedly destroying one's own life. A word of caution and exclusion: at law a suicide while insane is an accident and so also is an unprovoked homicide.[2] No attempt will be made here to distinguish between sane and insane suicides, thus limiting the discussion to distinctions between accidents and suicides, as those terms are commonly understood and defined.

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† Well-known and authoritative general sources, both legal and medical, are listed at the end of the paper under Additional References. Footnotes giving the author's last name with a page citation will refer the reader to the Additional References for the title, edition, year of publication, and publisher.

[1] Most texts on medical jurisprudence have introductory chapters giving good advice concerning the investigation which should be conducted at the scene of violence and upon postmortem examination.

[2] Cornelius, pp. 52–62. Yet if Lothario should disregard threats and call unarmed upon the irate husband, a jury might call it suicide! Central Nat. Bank v. Genrl. Amer. Life Ins. Co. (C.C.A. 6, Ohio, unreported) 11 CCH Life Cases 241, s.c. 105 F. (2d) 878 (1939), s.c. 136 F. (2d) 821 (1943).

II

SUBSTANTIVE LAW—HOW THE ISSUE ARISES

A

Historical. Miscellaneous Problems at Criminal and Civil Law

1. At Criminal Law

Until a modern and enlightened social philosophy fostered insurance for accidental injuries or death arising at work or elsewhere, suicide presented very few problems except those which concerned the King and the Church, criminal and ecclesiastical law. Their attitude of aversion and condemnation left a very definite impression upon the common law, although in modern times a cynical civilization which has created a richer and more desirable life has, paradoxically, relaxed many restraints, and, in fact, created a few more incentives for the voluntary passage to "the undiscovered country from whose bourn no traveler returns."

In ancient times suicide was not frowned upon with disfavor by the Church.[3] Neither Moses nor Christ spoke of it. When the deed was mentioned it was detailed as a historical fact without condemnation. It was not a crime except when committed by soldiers who thereby weakened the army and encouraged cowardice in battle.

During the Middle Ages and until only a few centuries ago suicide was considered an offense so heinous in England that the law pursued it after death, and to punish it inflicted degradation upon the body of the deceased. A stake was driven through the body and it was buried in the public highway. All of the deceased's estate was forfeited to the crown, and his soul and family lost many ecclesiastical solaces and beneficences. The sternness of some of these penalties was ameliorated in 1823 when burial without Christian rites was permitted in any parish graveyard between the hours of 9 and 12 at night. An Act of Parliament passed during the reign of Henry III providing for confiscation of the property of a suicide was not repealed until 1870. By an Act passed in 1882 all restrictions upon burials of suicides were finally lifted, except that Christian rites could not be conducted unless the suicide was *non compos mentis* at the time.[4] Some sects still refuse a Christian funeral to a suicide, either by rites or burial in hallowed ground.

It is only natural that many good folk recoiled from these barbaric penalties and sought means to avoid them. For many decades in England one could seldom find a coroner's jury returning a suicide verdict without tacking on the excupatory phrase, "while suffering from a temporary mental

[3] Bunzel, Bessie: Suicide, article in Encyclopedia of Social Sciences, 1934, vol. 14, pp. 454–459 with selective bibliography of authoritative books on suicide and its problems. A more comprehensive treatment of the subject with a longer bibliography may be found in Dublin, L. I., and Bunzel, Bessie: To Be or Not to Be, N. Y., Harrison Smith and Robert Haas, 1933.

[4] Bunzel, Bessie, op. cit., supra, f. n. 3; Halsbury's Laws of England, ed. 2, London, Butterworth & Co., 1933, vol. 9, p. 455.

aberration," for the poor unfortunate suicide who took his life while bereft of reason was not disgraced or punished.[5] If it was certain that the man was sane when he murdered himself, the jury could always disregard the evidence and find that he *accidentally* hanged himself or fired a ball into the roof of his mouth. These convenient mechanisms of escape continue in modern favor, not only in coroners' inquests,[6] but in civil suits where a suicide may carry with it a financial reward from an employer or an insurance company.[7]

In some states and in England it is a crime to attempt to commit suicide[8] or to aid or abet another by pact or otherwise in so doing,[9] and in such instances the prosecution must prove that the act was suicidal and not accidental. Quite often a murderer will attempt to conceal his crime by arranging the scene as a suicide. If the active agency of the murderer is established, he may attempt to mitigate the offense or excuse his conduct by claiming that he merely assisted the deceased to commit suicide.[10] In a criminal prosecution the State may show that the accused attempted to commit suicide as a confession of guilt.

2. *At Civil Law*

There are not many occasions at law, other than those to be mentioned under B, C and D, infra, where it becomes necessary to establish whether

[5] Blackstone, writing in the 18th century said: "The party must be of years of discretion, and in his senses, else it is no crime. But this excuse ought not to be strained to that length to which our coroners' juries are apt to carry it, viz.: that the very act of suicide is an evidence of insanity; as if every man, who acts contrary to reason, had no reason at all: for the same argument would prove every criminal *non compos*, as well as the self-murderer. The law very rationally judges that every melancholy or hypochondriac fit does not deprive a man of the capacity of discerning right from wrong; which is necessary, as was observed in a former chapter, to form a legal excuse. And therefore if a real lunatic kills himself in a lucid interval, he is a *felo-de-se* as much as another man." 4 Blackstone Comm. 189. What Blackstone thus said almost 200 years ago in England is a succinct statement of the law today in the overwhelming majority of the United States.

[6] In England, where a sane suicide is a crime, coroners' inquests in 4,846 suicides in 1928 returned the verdict of *"felo-de-se"* (sane suicide) in only 88 cases. East, W. Norwood: Suicide from the medicolegal aspect, British Med. Jr., 1931, ii, 241.

[7] Nor should juries bear all of the condemnation. The corruptibility and incompetence of some coroners have led to much criticism and a substantial movement toward the medical examiners system. See abstracts from several reports on coroners' laws in Herzog, p. 7 ff. Judges, too, have contributed their full share to the avoidance of a verdict of suicide. It was the legal mind and not a statistical, scientific analysis of facts and probabilities that created the presumption against suicide, endowed it with the character of evidence, instructed juries that they might consider it as such, and so used it procedurally as to shift the burden of proof to a party who should bear no such onus. Limitations of space and subject forbid our demonstration of the fallacy of this presumption which has been blindly accepted, as the law so often does, merely because other courts have previously done so. Having served a questionable purpose during a dead age, viz., the avoidance of penalties created by the stern hand of authority and destroyed by the very arm of law supposed to enforce them, this vestigial survival, the presumption against suicide, should be exposed and excised from the corpus juris.

[8] 92 A. L. R. 1180; 13 R. C. L. 720; 60 C. J. 997. Insanity is a defense (f. n. 5, supra) but drunkenness is no excuse, though it is a fact to be considered in determining whether an accused intended to commit suicide. Rex v. Doody [1854], 6 Cox, C. C. 463; Rex v. Moore [1852], 3 Car. & Kir. 319.

[9] 13 A. L. R. 1259; 60 C. J. 998.

[10] Thus, if the accused hanged his wife, it would be murder; if he assisted her to hang herself, it would be manslaughter. State v. Ludwig, 70 Mo. 412 (1879). If she hanged herself, it would be suicide, a complete defense.

a suicide was committed or an attempt thereat made. Most workmen's compensation laws provide benefits for accidental injuries and death arising out of and in the course of an employment, and exclude liability for death or disability due to injuries intentionally self-inflicted.[11] Under almost all compensation laws an insane suicide resulting from a compensable accidental injury is an accidental death; a sane suicide is not.[12]

A hospital or asylum or other person charged with the care of the physically or mentally ill may be liable in damages for the negligent failure to prevent a suicide.[13] Occasionally, a person sued for damages because of injuries or death alleged to have resulted from negligence will raise the defense that such damages arose from a suicide attempt.

A few states have dramshop and civil damages acts rendering one liable for damages or death caused by the negligent sale of intoxicating liquors, the use of which by the purchaser has caused death or injury to himself or another.[14] It is well known that an alarming percentage of suicides are committed either directly or indirectly as a result of acute or chronic alcoholism,[15] so that in civil suits for damages for violation of such statutes it may become important to determine whether a death or injury was caused by suicide or accident.

There is a conflict in authority whether one who tortiously injures another is liable for subsequent injuries or death caused by a successful or unsuccessful suicide which resulted from unendurable pain, despondency or mental aberration arising from the first injury.[16] In those jurisdictions where self-destruction is a crime, a maliciously false statement that a person committed suicide may be punishable at criminal law, but does not render one civilly liable to the surviving next of kin, unless, perhaps, in such states as Missouri, where a libel, by statutory definition, includes "any malicious

[11] Schneider, William R.: Workmen's Compensation Laws, perm. ed. 1943, vol. 4, p. 4442. For illustrative cases where the issue was accident or suicide, see 143 A. L. R. 1227.

[12] 56 A. L. R. 459; 143 A. L. R. 1227; 12 N. C. C. A. (N. S.) 298. See also f. n. 16, infra.

[13] 7 N. C. C. A. 82, 88; 10 N. C. C. A. 749; 36 N. C. C. A. 618; 23 A. L. R. 1277.

[14] 9 N. C. C. A. (N. S.) 176, 192–200; 23 A. L. R. 1276. As to liability of a druggist for furnishing the means to commit suicide, see 11 N. C. C. A. (N. S.) 752.

[15] This fact, casually noted from a reading of a considerable number of court opinions, seems to have adequate statistical support. It has been reported that out of 1000 consecutive cases of attempted suicide admitted to Brixton prison in England, the major cause or motivation in 141 was "alcoholic impulse with amnesia," in 171 was "alcoholic impulse—memory retained," and in 31 was "post-alcoholic depression," a total of over one-third of the entire group. These figures do not include instances of insanity or other mental states due in whole or in part to alcohol. Nor do they include those cases where the suicide by the use of alcohol is finally able to screw his courage to the sticking point to carry out self-destruction premeditated for other reasons. East, W. Norwood: Medical Aspects of Crime, 1936, chapter V, p. 141. In another study of 1000 attempted suicides, abnormal mental states were found upon admission to a hospital in 23 per cent of the cases. In more than half of these there was a diagnosis of acute or chronic alcoholism. Lendrum, F. C.: A thousand cases of attempted suicide, Am. Jr. Psychiat., 1933, xiii, 479. Menninger believes that addiction to alcohol is a form of suicide. Menninger, Karl A.: Man Against Himself, 1938, Harcourt, Brace & Co., N. Y., chap 3. A readable recent book with a good bibliography of assistance both to the student and casual inquirer is Haggard, H. W. and Jellinek, E. M.: Alcohol Explored, 1945, Doubleday-Doran & Co., N. Y.

[16] 8 N. C. C. A. 1025; 23 A. L. R. 1273; 79 A. L. R. 370, and see f. n. 12, supra.

defamation . . . designed to blacken and vilify the memory of one who is dead, and tending to scandalize or provoke his surviving relatives and friends." [17]

In some jurisdictions a will or gift made in contemplation of suicide is void if it can be proved that death resulted from suicide rather than accident or some other cause.[18]

B

Coroners' Inquests and Death Certificates

One of the most important duties of every coroner and medical examiner is the determination of whether a death was caused by disease, accident, suicide or homicide. The result of that determination is usually made a part of the death certificate required by statute. The coroner's verdict or the death certificate is often incorporated in proofs required by insurance companies. Under many statutes, the attending physician is required to make out the death certificate even though there is an inquest or autopsy. Most statutes relating to death certificates require the signer to certify whether the death was "probably" due to accident, suicide or homicide. Insurance companies usually ask the attending physician to sign one of the proofs. These certificates and proofs, prepared by coroners and attending physicians, are in many jurisdictions admitted in evidence as prima facie proof of the cause of death, rebutting the presumption against suicide, and binding and conclusive unless contradicted or explained.[19]

C

Life Insurance

Suicide as a Theory of Recovery. If an insured disappears and remains unheard from, his whereabouts unknown, under circumstances which do not indicate that he has died, the beneficiary must wait seven years before his insurance may be collected, for it is not until that time that a rebuttable presumption arises that the insured is dead. Therefore, it is to the interest

[17] R. S. Mo. 1939, sec. 4758. Compare: Hughes v. New England Newspaper Pub. Co., 312 Mass. 178, 43 N. E. (2d) 657 (1942), and annotation in 146 A. L. R. 739, "Civil Liability for Defamation of the Dead." In a recent case dealing with privileged communications to a physician, the court said: "Unless the circumstances leading to suicide are in themselves immoral or disgraceful, the mere act of self-destruction, of itself, does not necessarily tend to disgrace the memory of the decedent." Bolts v. Union Central Life Ins. Co. (City Ct.) 20 N. Y. S. (2d) 675 (1940).

[18] 35 Yale Law J. 379; 30 Mich. Law R. 626; 32 Col. Law R. 710.

[19] 93 A. L. R. 1342 (conclusiveness of proofs); Cooley, vol. 6, pp. 5466–5468, 5477–5478; Couch, vol. 8, sec. 2200 p. 7111 ff., secs. 2225–2229, p. 7213 ff. As to whether the presumption against suicide is overcome by a death certificate, coroner's verdict or similar documentary evidence, see 159 A. L. R. 171. For admissibility of death certificates and all of the statements therein contained, see f. n. 72, infra. In one case a sheriff summoned a local doctor to view a drowned body unknown to either of them. "I guess it is another suicide," said the doctor and so signed the death certificate. The insurance company disclaimed liability and the widow was forced to file suit. "At trial both the sheriff and the doctor abjectly confessed themselves in error for the casual way they did their business." Eastern Commercial Trav. Acc. Assoc. v. Sanders (C. C. A. 1, Mass.) 108 F. (2d) 643 (1940).

of the beneficiary to show that the insured disappeared under circumstances pointing to accidental death, suicide or homicide while the policy was in force. Such circumstances may be sufficient to raise an inference of death in less than seven years, or may reinforce the claim at the end of the presumptive period of seven years.

Suicide as a Defense.[20] In the absence of a statute or policy provision, suicide while sane in some jurisdictions is a defense to a life policy payable to the insured's estate, but is not a defense if the policy is payable to a third person as beneficiary, unless, however, the policy was fraudulently procured with the intention to commit suicide and thus create an estate for the beneficiary.

Unless excluded by the policy, a suicide while insane is never a defense to a life policy.

Therefore, many life policies expressly exclude liability for death by suicide. This clause will avoid payment for sane suicides, but not insane suicides, unless the exclusion reads "suicide, sane or insane," and even then a few states will allow a recovery where the insured was so insane that he did not understand the physical consequences of his act and did not intend to end his life.[21]

Although most life policies do contain the "suicide, sane or insane" exclusion, they usually have so-called incontestable clauses providing that the defense of suicide may not be raised after a certain period, usually one or two years after the policy was issued.

D

Accidental Death and Disability Benefits

Accident Insurance and Double Indemnity.[22] As mentioned above, an insane suicide and an unprovoked homicide are accidents, whereas sane suicide and provoked homicides, where the insured is the aggressor, are not accidents. Therefore, if a policy in its insuring clauses covers accidental deaths, the insurer is liable for an insane suicide or an unprovoked homicide, but not for a sane suicide.

Most accident policies with death benefits and most life policies with double indemnity provisions expressly re-define the coverage by excluding suicide, but this will not eliminate insane suicides unless the clause, as it usually does, reads "suicide, sane or insane." Many such policies also exclude liability for a death from injuries intentionally inflicted by another person.

[20] The general rules laid down in this and the next section of this paper dealing with accident insurance are so well-established that a general citation to several authoritative sources will suffice for our purposes. However, many exceptions and conflicts will be found. See: Appleman, vol. 1, chapter 19, pp. 419–451; Cooley, vol. 6, pp. 5363–5376, 5397–5489; Couch, vol. 5, pp. 4020–4022, vol. 6, pp. 4611–4667; Joyce, vol. 4, chapter LXXI, pp. 4407–4480; 37 C. J. 551–556; 29 Am. Jur. 697–704.

[21] 153 A. L. R. 801; 138 A. L. R. 827; 35 A. L. R. 165.

[22] See f. n. 20, supra, and, in addition 1 C. J. 443–444 and Cornelius, pp. 52–62.

In Missouri, a statute invalidates the policy exclusion of insane suicides, but by judicial construction does not affect the rule that a sane suicide is not an accident.[23]

It is not unusual to find policies covering accidental deaths bearing other exclusions collaterally affecting death by suicide and avoiding the problem of distinguishing between accident, suicide (sane or insane), homicide, and, in some cases, disease.[24] Thus, a policy may, by exclusion, eliminate coverage for certain kinds of deaths whether due to accident, disease, homicide or suicide. The policy may exclude deaths due "directly or indirectly, in whole or in part," to disease, bodily or mental infirmity, infections (of certain types), medical treatment, pregnancy, hernia, freezing, sunstroke, drowning, gas, poison, gun-shot wounds, the influence of narcotics or liquor, war, aviation, violation of law, voluntary exposure to unnecessary danger, etc.

Disability Benefits. If an insured unsuccessfully attempts to commit suicide and suffers a loss of a member or disability for which, if accidental or due to disease, he would be entitled to benefits under an insurance policy, he is not entitled to such benefits even in absence of an exclusion in the contract, unless he was insane at the time of the attempt.[25] In these cases, as in all of those heretofore mentioned, it will be necessary to determine whether the act was accidental or suicidal.

III

PROCEDURAL LAW—HOW THE ISSUE IS DECIDED[26]

A

Pleading, Burden of Proof and Presumptions[27]

As a general rule it is the duty of the party who asserts a fact necessary to his cause of action or affirmative defense to plead and prove it. Therefore, in a suit on a *life* insurance policy where suicide is an affirmative de-

[23] The Missouri statute barring suicide as a defense was for many years misconstrued to make a sane suicide an accident! Missouri became a mecca for suicides. For a while there was a veritable flurry of suicides on street cars and railroad trains, since a sane suicide not only permitted the beneficiary to collect double indemnity, but, under some policies covering accidents on public conveyances, triple indemnity as well!

[24] Such exclusions are fully discussed in Appleman, vol. 1, chapters 22–32; Cooley, vol. 6, pp. 5293–5397; Couch, vol. 6, chapter 17; Joyce, vol. 4, chapter LXX.

[25] There may be some conflict on the question. Elwood v. New England Mut. Life Ins. Co., 305 Pa. 505, 158 Atl. 257 (1931); compare Prudential Ins. Co. v. Rice, 222 Ind. 231, 52 N. E. (2d) 624 (1944). See 49 U. of Pa. Law R. 1365.

[26] Here will be found some of the reasons so many apparently unjust jury verdicts have been permitted to stand. Here is drawn the heavy line separating the province of the court from that of the jury. Until the judicial process is understood it cannot be criticized or even materially assisted by the investigator. The layman or physician should not be too quick to find fault with these rules, for they are the very basis of the jury system, and for the most part and with very few exceptions have had the support of jurists for years in all states.

[27] "Perhaps no topic of the law has perplexed the courts more than the scope and effect of legal presumptions. The complexities and subtleties of the subject, enhanced no doubt by the variant theories regarding them and the confusion of thought manifested in the discussion of the questions incident to them, have given rise to a condition that has been characterized as a 'welter of loose language and discordant decisions.'" Tyrrell v. Prudential Ins. Co., 109 Vt. 6, 192 Atl. 184 (1937).

fense, the burden of proof is on the defendant to plead and prove that the insured committed suicide. On the other hand, in a suit on an *accident* insurance policy *or* on a life policy for *double indemnity,* the defendant is not required to plead or prove suicide as a defense since the plaintiff has the burden of pleading and proving that death resulted from a cause within the coverage, accident, so that the defendant by merely denying the plaintiff's assertion may prove any fact, such as suicide, which tends to disprove the plaintiff's theory of accident. The failure to distinguish a suit on a life policy from one for accidental death benefits (double indemnity or accident policy) has introduced much conflict, misunderstanding, confusion and injustice into the trial and decision of *accident* insurance cases.[28]

The phrase "burden of proof" means "the risk of non-persuasion" or the duty of affirmatively proving one's case or defense, so that if there is no substantial evidence in support thereof, the issue cannot be submitted to the jury, but must be ruled out of the case as a matter of law by the judge. If there is substantial evidence warranting the submission of the issue to the jury, then the party carrying the burden of proof must establish his case to the satisfaction of the jury by a preponderance or greater weight of the credible evidence.

The burden of proof never shifts, but continues to abide throughout the trial with the party upon whom it was originally cast. However, the burden of going forward with the evidence does shift, so that after one litigant has made out a prima facie case and thus, for the time, sustained his burden of proof, the duty of going forward with evidence to meet the prima facie case shifts to his adversary.

These rules are of considerable importance because it is almost universally accepted that as soon as there is proof that a death was violently met, the law presumes that the death was not caused by suicide or murder. Therefore, in a suit for accidental death benefits or for workmen's compensation, the beneficiary is said to make a prima facie case merely by proving that death was violent, and no further evidence is required unless that prima facie case is met by the defendant who has the burden of going forward with evidence (but not the burden of proof).

However, a presumption is not evidence; it is merely a rule of procedural law covering the duty of going forward with evidence. Once substantial evidence of the facts appears, whether introduced by the plaintiff or the defendant, or both, the presumption disappears from the case, has no probative

[28] In suits on *life* insurance policies where the evidence is entirely or mostly circumstantial, the defendant is required not only to produce substantial evidence of suicide but also to exclude every reasonable hypothesis of accident or homicide before it is entitled to a directed verdict as a matter of law. However, this rule does not apply in accident cases, since the defendant's failure to prove suicide as a matter of law does not relieve plaintiff of his burden of proving accident, so that if there is no substantial evidence of accident, or if the cause of death can only be determined by speculation, conjecture or surmise, then and in either such event the plaintiff has failed to carry his burden of proof and the defendant is entitled to a direct verdict. The insurer "is not required to eliminate every speculative, fantastic, conjectural, frivolous and imaginary hypothesis of death in any other way." N. Y. Life Ins. Co. v. Hunter, 60 Ariz. 416, 138 P. (2d) 414 (1943).

force, has no weight as evidence, and the jury cannot be instructed to consider it in arriving at a verdict.[29]

There are several other presumptions which the law occasionally recognizes and which may have some bearing on the decision of cases within the subject under consideration. There is a presumption that all men are sane and conscious of the consequences of their actions. Therefore, when the insured has committed suicide and plaintiff is relying upon the theory that he was insane and his death thus accidental, the burden of proving that the insured was insane at the time of the act rests upon the plaintiff.[30] The mere fact that the insured committed suicide raises no presumption of insanity so far as the court is concerned, although many laymen, physicians and psychiatrists think differently, much depending upon their definition of insanity.[31] On the other hand, proof that the insured was insane at the time of an unexplained violent death may destroy the presumption against suicide.[32] We again sound the warning note that all of these presumptions are not evidence though they may, temporarily and until rebutted, take the place of evidence.

B

ADMISSIBILITY OF EVIDENCE

Evidence is admissible in a trial if it is material, competent, relevant, and satisfies prescribed modes of proof. It is material and relevant if it tends to establish the truth of the fact sought to be proved, i.e., if it might be accepted by reasonable men as tending to prove the fact. The present tendency is to relax the rules of evidence and admit much evidence which was formerly withheld from a jury.

It is, of course, entirely proper to prove all of the facts and circumstances leading up to, surrounding and following the injury which tend to establish

[29] The sardonic comment of Judge Belt in Wyckoff v. Mutual Life Ins. Co. of N. Y., 173 Ore. 592, 147 P. (2d) 227 (1944), though unjustified, requires citation: "Some text writers, law professors and judges who have espoused the Wigmore doctrine have vied with one another in an effort to show how flimsy and unsubstantial a presumption of law really is. This 'phantom of the law' has been likened to 'bats flitting about in the twilight and then disappearing in the sunshine of actual facts,' and to a house of cards that topples over when rebutted by evidence. It remained for Professor Bohlen to head the class when he said a presumption of law was like Maeterlinck's bee which, after functioning, disappeared."

[30] Laventhal v. N. Y. Life Ins. Co. (D. C., Mo.) 40 F. Supp. 157 (1941) a case decided solely upon presumptions. It must be shown that the insured was insane at the time of the act, so that plaintiff may encounter some difficulty where the insured is subject to only temporary fits of insanity. Appleman, vol. 1, p. 436. Blackstone long ago recognized the fact and burden of proof, f. n. 5, supra.

[31] 37 C. J. 620; N. Y. Life Ins. Co. v. King (C. C. A. 8, Mo.) 93 F. (2d) 347 (1937). As to disqualifying jurors who believe so, see Edwards v. Business Men's Assur. Co., 350 Mo. 666, 168 S. W. (2d) 82 (1942). However, it has been held that suicide is evidence of insanity. Horvath v. N. Y. Life Ins. Co., 65 S. D. 480, 275 N. W. 258 (1937); Wigmore on Evidence, ed. 2, secs. 228, 2500, 2501. Despite this rule, it is also held that suicide does not overthrow the presumption of sanity.

[32] Cooley, vol. 6, pp. 5454–5459. "The presumption [against suicide] in the case of a sane man is based upon his sanity, and the fact of insanity being shown, the ground of the presumption is gone." Horvath v. N. Y. Life Ins. Co., 65 S. D. 480, 275 N. W. 258 (1937) and cases cited. The better rule is that insanity merely destroys the presumption against suicide and does not create any new presumption in favor of it. 112 A. L. R. 1278.

its cause and the motive or lack of motive for suicide at the time of its infliction. The courts are also very liberal in permitting proof of any fact which might prove or disprove the presence or absence of a motive for suicide.

There is almost no limit to the type of evidence developed by the ingenuity of litigants to prove or disprove theories of the cause of death. The best rules to guide the investigator are: (1) assume that the evidence is admissible; (2) gather, fix, and preserve it in such form that it will be available, useful, and not subject to contradiction months or years later when it may be needed; (3) obtain all of the evidence possible of a corroborative nature with special emphasis on the use of methods not subject to impeachment, i.e., the use of disinterested witnesses, signed statements, photographs, and scientific proof [33]; (4) do not neglect negative evidence such as written, signed statements that witnesses in a position to know a fact did not know it, because it is a continual source of chagrin to the lawyer to find witnesses, who orally stated to the investigator that they did not know a fact, present in court months later with vivid recollections of the most minute details.

C

Sufficiency of the Evidence

Although the jury is the sole judge of the credibility of the witnesses and the weight to be given to their testimony, it is the special province and duty of the court to determine, as a matter of law, whether there is sufficient substantial evidence in support of an issue from which a jury could find the facts in favor of the party with the burden of proof. In deciding this question of law, the court is obliged to view the evidence in that light most favorable to the party carrying the burden of proof, giving to him the benefit of all logical inferences which may be drawn from all of the evidence and rejecting all evidence opposed thereto, except that evidence of his adversary consistent with the proponent's testimony and not in conflict with his theory of recovery. After so considering the evidence, the issue is one for the jury and cannot be ruled by the court as a matter of law if the minds of reasonable men might differ as to the ultimate fact in issue. If the evidence is documentary

[33] A good illustration is Gilpin v. Aetna Life Ins. Co., 234 Mo. A. 566, 132 S. W. (2d) 686 (1939) where the deceased with adequate motive for suicide (syphilis, debts, involvement with another woman, etc.—all disputed) was found in his car with a revolver wound in his head under circumstances which the court held might indicate that death was either accidental or suicidal. Among other conflicts in the evidence were the existence, extent and location of powder marks, the distance the revolver must have been from the head when the shot was fired, the presence or absence of a bruise on the hand and, of all things, the location of the entrance wound itself. The coroner, a pathologist, said that it was above the right ear and that the path of the bullet was straight through the head. The court felt helpless to accept this evidence of a scientific, disinterested witness as conclusive, and said that it was for the jury to say whether such evidence should be rejected in favor of that given by friends of the deceased (and the beneficiary) who variously located the entrance wound in the middle of the forehead, over the right eye, and at the temple! A photograph would have avoided such conflicts resulting either from incompetence or dishonesty on the one side or the other. Photographs of corpses are admissible in evidence. 159 A. L. R. 1413.

or uncontested or the facts are agreed to, then the court may decide the issue in favor of the plaintiff as a matter of law. The court may be convinced that the preponderance or weight of the evidence is in favor of one party, but, except in a few states, it may not determine the case on its view of the weight of the evidence so long as there is sufficient substantial evidence to the contrary. In such case, the jury alone is privileged to decide the issue.

Thus, if the widow-beneficiary testifies that her husband was happy and had no domestic or financial troubles, it is the privilege of the jury to believe her and reject the positive contrary testimony of ten witnesses who testify for the insurance company. The coroner's physician may testify to the location of wounds and presence or absence of powder burns, but an interested relative of the deceased may testify and be believed to the contrary. When photographs and documentary evidence are introduced, suicide notes, for instance, the court and jury both are usually inclined to accept such evidence as establishing the facts, unless they are explained or in some way weakened or impaired.

These observations indicate how important it is to "clinch" a fact beyond any dispute at the time of the original investigation. Let no one *suppose* that *his* word will be accepted over that of any other man when both stand before a jury. And let there be no one so naive as to believe that witnesses are never mistaken or dishonest. There are often strong motives and virtually no real deterrants to perjury, and even where testimony is not knowingly false, the witness may be mistaken and do equal damage. There is no fact immune from rebuttal by dishonesty, ignorance or both. Caveat investigator!

In recent years most courts have become more impartial in the administration of insurance law. More stress is laid on common sense and sound scientific testimony than on artificial presumptions and rules of law. With an understanding of the burden of proof and effect of presumptions there is an increasing tendency to return to three fundamental principles applied in all other cases: *first,* evidence must be substantial before a jury is allowed to accept it as proof of the ultimate fact; *second,* speculative possibilities are not evidence and the jury should not be permitted to find a verdict by mere speculation and conjecture[34]; and, *third,* where under the evidence an injury

[34] A "fanciful theory of accident" cannot be spun out on bare possibilities since verdicts must rest upon probabilities. Love v. N. Y. Life Ins. Co. (C. C. A. 5, Miss.) 64 F. (2d) 829 (1933). "We do not think these theories can be accepted with any show of reason, or that they would be seriously considered if this were not a controversy between a bereaved widow and an insurance company." Brotherhood of Maintenance of Way Employees v. Page, 197 Ark. 498, 123 S. W. (2d) 536 (1939). "Jurors are not permitted to shut their eyes to what everybody else sees and understands and wander off into fields of imagination and suspicion in order to reach verdicts. Courts are more and more realizing and declaring that they must not permit themselves to be more ignorant than anybody else or fail to see what is plain to everyone and everybody except a court." Deweese v. Sovereign Camp, W. O. W., 110 Kan. 434, 204 Pac. 526 (1922). This result is, as indicated, largely chargeable to erroneous concepts of the burden of proof and the presumption against suicide, so that too frequently "judge and jury alike have been unable to take a common sense view of the facts of life, and have seemed to be the only persons in the community who did not clearly understand what had taken place." Jefferson Standard Life Ins. Co. v. Clemmer (C. C. A. 4, Va.) 79 F. (2d) 724 (1935).

or death might or could have resulted from either one of two causes for only one of which the defendant would be liable, and the plaintiff has not sustained his burden of proving that such cause was the more probable, then no recovery may be had.[35]

IV

EXPERT AND NON-EXPERT EVIDENCE OF PROBATIVE FORCE

Suicide is death from (1) injuries self-inflicted (2) with intention to end one's life. Accidental injuries may or may not be self-inflicted; they are usually caused by some accidental means external to the individual; they are, of course, never inflicted with intention to end one's life. Therefore, in seeking evidence of probative force to distinguish between accident and suicide, we naturally turn to two sources: (A) External Evidence, to determine whether the injury was self-inflicted, and (B) Internal Evidence, to determine if there was an intention to end one's life. This evidence may be direct or circumstantial. According to Blackstone, "evidence signifies that which demonstrates, makes clear, or ascertains the truth of the very fact or point in issue, either on the one side or the other." [36] "Direct or positive evidence is to the precise point in issue, as in the case of a homicide, that the witness saw the accused inflict the blow." [37] In a suicide case the testimony of an eye-witness, a suicide note or other declaration of intent, and an unquestioned photograph may be termed direct evidence, and this is important because in such cases there is no need to resort to circumstantial evidence or lay stress on motive.[38] "Circumstantial evidence is that which relates to a series of other facts than the fact in issue, which by experience have been found so associated with that fact that in the relation of cause and effect they lead to a satisfactory conclusion." [39] The distinction is of some importance because of the frequent insistence that where evidence is circumstantial it must negate every reasonable hypothesis of death by accident.[40] This, as we

[35] "Where proved facts give equal support to each of two inconsistent inferences neither is established, and judgment must go against the party on whom rests the necessity of sustaining one of them against the other." N. Y. Life Ins. Co. v. Ittner, 64 Ga. A. 806, 14 S. E. (2d) 203 (1941), dissent by Felton, J., citing cases; N. Y. Life Ins. Co. v. Prejean (C. C. A. 5, La.) 149 F. (2d) 114 (1945). "There were no facts or circumstances from which the jury could infer legitimately to the exclusion of other inferences equally plausible that the insured's death resulted from accident." Waldron v. Met. Life Ins. Co., 347 Pa. 257, 31 A. (2d) 902 (1943). "In such circumstances, under contradicted evidence, the party having the burden of proof cannot prevail." Christensen v. New Eng. Mut. Life Ins. Co., 71 Ga. A. 393, 31 S. E. (2d) 214 (1944), s. c. 197 Ga. 807, 30 S. E. (2d) 471, s.c. (unreported Ga. A.) 9 CCH Life Cases 268. Of course, if there is no substantial evidence of accident, then the issue of accident cannot be submitted to the jury and it is immaterial where the burden of proof lies in a suit for accidental death benefits. Fox v. Mut. Ben. H. & A. Assoc., 61 Ga. A. 835, 7 S. E. (2d) 403 (1940).

[36] 3 Blackstone Comm. 367.

[37] Jones, vol. 1, p. 16.

[38] Webster v. N. Y. Life Ins. Co., 160 La. 854, 107 So. 599 (1926), a learned opinion frequently cited and followed.

[39] Jones, vol. 1, p. 16.

[40] Cooley, vol. 6, p. 5473; Gilpin v. Aetna Life Ins. Co., 234 Mo. A. 566, 132 S. W. (2d) 686 (1939); N. Y. Life Ins. Co. v. Satcher, 152 Fla. 411, 12 So. (2d) 108 (1943).

have demonstrated, may be true in criminal cases, but it is not true in civil cases.[41]

Once all of these rules of law are understood, we are ready to discuss evidential problems in distinguishing accident from suicide. "But that is a question of fact, because, like the question of what is the proximate cause of an injury, it is not a question of science or legal knowledge, but each case must necessarily stand on its own probative facts and circumstances," and no one case can serve as a complete precedent and be decisive of any other.[42]

A

External Evidence: Was the Injury Self-Inflicted?

First, determine the physical cause of death.

Was it due to natural or unnatural causes: disease, poison, gun-shot wound, gas, drowning, a fall or impact? Where there are multiple wounds as possible causes of death, one must decide which injury was inflicted first, whether it alone was sufficient to cause death, whether the other injuries could have been accidentally or self-inflicted thereafter, and what diseases and wounds and other causes actually contributed to bring about death. Burning may be used to conceal a suicide or homicide. A determination of the concentration of alcohol in the blood may help to prove that the burning resulted from an alcoholic stupor.[43] A cerebral hemorrhage or coronary occlusion may precede and cause a fall or auto accident, which in turn may cause death,[44] or death may result from the hemorrhage or clot despite extensive injuries suffered in the fall. If a body is recovered from water it must be determined, if possible, whether death resulted from drowning or whether it preceded immersion and was caused by disease, suicide or homicide.[45] Carbon monoxide determinations should readily disclose whether death occurred as a result of inhalation of that gas where the body was found

[41] Cox v. Met. Life Ins. Co., 139 Me. 167, 28 A. (2d) 143 (1943). See also: Mut. Life Ins. Co. of N. Y. v. Hamilton (C. C. A. 5, Fla.), 143 F. (2d) 726 (1944).

[42] Webster v. N. Y. Life Ins. Co., 160 La. 854, 107 So. 599 (1926). The court did not mean to say that scientific knowledge and proof would not be evidence. It meant that "the entire question is one of fact, and not one of law." Soecker v. Met. Life Ins. Co., 51 Cal. A. (2d) 479, 125 P. (2d) 105 (1942). See also f. n. 74, infra.

[43] Jetter, Walter W.: When is death caused or contributed to by acute alcoholism?, Clinics, 1943, i, 1487. The presence or absence of alcohol in the blood in a sufficiently high concentration may make accident plausible or "may account for suicidal dementia or for behavior changes likely to provoke assault." Moritz, Alan R. and Lund, Herbert: Special evidentiary objectives of the medicolegal autopsy, Jr. Tech. Meth. and Bull. Internat. Assoc. Med. Mus., 1943, No. xxiii, p. 71. The blood alcohol determination may assist the beneficiary in showing that the death was accidental; or in any case of death by burning or other cause, the insurer may wish to establish the fact in order to invoke provisions of the policy excluding liability if death occurred while the insured was intoxicated or under the influence of liquor. It is usually not necessary to show that there was a causal connection between the condition and the death. Appleman, vol. 1, sec. 465–582. As to admissibility and weight of evidence based on scientific tests for intoxication or the presence of alcohol in the system, see 127 A. L. R. 1513; 159 A. L. R. 209; 29 Va. L. Rev. 749 (1943).

[44] Such a death is usually held to have been effected by accidental means, but there is no liability in most states if the policy further provides that it will not cover a loss resulting from or caused directly or indirectly or in whole or in part by disease. Cornelius, pp. 42–50.

[45] Kahn v. Met. Life Ins. Co. (Mo. Sup.) 240 S. W. 793 (1922), where there was evidence that the death might have been caused by a heart attack, either alone or causing a fall from a boat, or by poison or by drowning or a combination of those causes.

in a garage, a gas-filled room or burning building.[46] Since almost all hangings are suicidal and not accidental or homicidal, one should be certain that the body was not strung up after a homicidal death to conceal the crime. Deaths from poison are the most difficult properly to prove in a court of law. There are few toxicologists who can fully qualify in that field. The usual hospital or coroner's autopsy generally obtains nothing more than the barest presumptive evidence of death from poisoning. A carefully conducted cross-examination can in almost every case destroy or seriously impair an opinion that death resulted from poisoning.

Second, note the location, size, direction or course, nature and extent of all wounds or other evidence of the body's reaction to the lethal agency. This would also include any evidence on the body, clothing or surroundings furnishing any clue to the nature of the weapon or agency causing death and its position before, at the time, and after it acted upon the deceased. Specific evidence in various types of violent deaths will be dealt with in division V of this paper.

Medical jurists have evolved many *general* rules, most of them of little practical value, for distinguishing accidental from suicidal or homicidal wounds and deaths. All of such general rules about the "usual" case have not one but many exceptions, and no one bit of evidence should alone decide the issue. Moreover, some of these rules or "clues" have more value than others in one case, but their *relative* importance may change in another case.

Illustrative of the numerous physical circumstances stressed by courts, which may furnish external evidence of assistance to the jury are: the time, place and circumstances of the death, the nature of the death wound, the opportunity which deceased had to kill himself at other times, the position of the body, the location of a bullet or other wound, the course and depth of the wound, the presence or absence of powder marks or singeing, the relative location of the instrument employed to take life, the presence or absence of evidence of violence or an out-cry, etc.[47]

Third, determine the source of the lethal agency, if any, which caused the death, the deceased's access to it at the time when the injury was presumably inflicted, and, finally, the physical relation of the agency to the deceased.

Fourth, reconstruct the events taking place when the injury occurred.

This is exactly what the jury will be thinking of from the opening moments of a trial. Is there substantial circumstantial evidence from which a logical conclusion can be reached, or must the investigator speculate and

[46] Gettler, A. O. and Freimuth, H. C.: The carbon monoxide content of blood under various conditions, Am. Jr. Clin. Path., 1940, x, 603; Merkel, H.: Findings of diagnostic value on burnt and charred bodies, Deutsch. Ztschr. f. d. ges. ger. Med., 1931, xviii, 232 (abstracted in Arch. Path., 1932, xiv, 425). Where a body is found burned or exposed to carbon monoxide and there is no significant concentration of that gas in the blood, the person died from other causes than burning or carbon monoxide poisoning before he was exposed to any significant amount of carbon monoxide. The blood within the heart or major vessels of a dead body will not absorb carbon monoxide from the air, but the peripheral parts of the body may absorb enough of the gas to give a so-called "characteristic" cherry-red coloring and thus mislead the casual viewer.

[47] 17 Amer. & Eng. Anno. Cas. 35–37; Ann. Cas. 1913 C, 1260.

guess in order to reconstruct the event? Is all of the evidence consistent with accident or suicide, or both?

B

Internal Evidence: Was the Injury Intentional?

Wigmore has said that "the evidential data available to prove the doing of any human act fall always into three groups, viz., prospectant, concomitant and subsequent." [48] We have borrowed his classification in dealing with the issue of whether an injury was intentionally self-inflicted.

1. Prospectant Evidence

Incentives and Deterrents. The proponent of the theory of accident will invariably show that the deceased had no reason or motive to commit suicide, and that a number of circumstances combined to deter him from such an act. The opponent, urging suicide, will attempt to show that such deterrents were lost or weakened and that the deceased had every incentive to abandon life and seek death by the escape mechanism of suicide. It is just as important for the beneficiary of an accident policy to show the absence of motive and the presence of natural deterrents as it is for the insurer to prove the contrary to be true.[49] However, it is not necessary to the theory of suicide that a motive be found.[50] The springs of human action are often hidden and of such obscure origin that not even a psychiatrist with the full voluntary cooperation of his patient can find them.[51] Some suicides are committed for ulterior motives never discovered,[52] and some occur not for any deep-lying or long-existing reason, but for shallow reasons as a result of sudden impulse

[48] Wigmore, John H.: Circumstantial evidence in poisoning cases, Clinics, 1943, i, 1507.

[49] "Motive is important and ofttimes its existence or lack of existence turns the scales against or in favor of the theory of suicide." Bayles v. Jefferson Standard Life Ins. Co. (La. A.), 148 So. 465 (1933). The beneficiary will attempt to show any one or more of the following facts which have been said to indicate that the death may be accidental: youth, good health, good habits, sobriety, industriousness, religious inclinations, a merry, cheerful disposition, "slept and ate well," good spirits, happy home life, kindness and affection to children, enjoyment of friends and genial companions, freedom from debt, satisfactory employment, etc. The insurer will look for evidence to the contrary. 17 Amer. & Eng. Anno. Cas. 38–39.

[50] A motive helps to decide the case, but is not necessary to a finding of suicide. N. Y. Life Ins. Co. v. Sparkman (C. C. A. 5, Fla.), 101 F. (2d) 484 (1939). "Motive was not an essential element requiring either proof on the part of the plaintiff or disproof on the part of the defendant." Leahy v. Travelers Ins. Co. (D. C., Ohio), 42 F. Supp. 26 (1941).

[51] "It is no reflection upon the profession of psychiatry to say that it necessarily deals in a field of conjecture. Even in the diagnosis of actual insanity, cases are rare in which trained psychiatric witnesses do not come to opposite conclusions. The opinions here relate to neurosis, a condition short of insanity, on which there are countless theories and infinite diagnostic possibilities. It is difficult to conceive of records in which the right of cross-examination is more important than the conjectures of a psychiatrist on a psychoneurotic condition." N. Y. Life Ins. Co. v. Taylor (C. C. A., Dist. Col.), 147 F. (2d) 297 (1944), passing on the admissibility of hearsay evidence and opinions contained in hospital records.

[52] The trial of John Thomson in 1857 for the murder of Agnes Montgomery at Eaglesham, near Glasgow, was the first case in Scotland of murder by prussic acid. The defense attempted to show that there was no motive for the deed. The Lord Justice Clerk, John Hope, instructed the jury: "It is a rash thing to attempt to set a bound to man's malignity, or to suppose that, because your honest and innocent hearts cannot enter into the motive of one committing such a crime, guilt must be excluded. We know not the depths of the depravity and malignity of the human heart, and numbers of desperate criminals would escape if one were to test things in this way, and to lay aside evidence of matters of fact from our inability to understand what led the man to do the deed." 27 Jur. Rev. 76 (1915).

and post-alcoholic and disease states unknown before death and undiscovered thereafter.[53] Then again, after a suicide, the family and friends are reluctant to reveal any facts which would reflect on them or the memory of the deceased. An opportunity to collect money from an employer or an insurer furnishes an additional reason for reticence.

The usual deterrents and incentives to suicide may be found by reference to age, domestic relations, financial circumstances, lack of employment, fear of arrest or imprisonment because of embezzlement or any other crime, physical and mental health (including acute and chronic alcoholism and post-alcoholic and disease states), frustrated love, religion, superstitions, philosophy and attitude toward life and death, etc.[54]

Knowledge of the Lethal Agency. It should be determined whether or not the deceased knew that the deadly agency which caused his death was capable of that effect. There could be no intention to end life unless the deceased knew the existence, presence or imminence of the thing which took his life and its deadly character. Acts preparatory to the deed may disclose the knowledge and intention requisite to proof of suicide, or the absence of such knowledge.[55]

Previous Attempts. Great stress is and should be laid upon the discovery that the deceased had previously attempted to commit suicide. Such attempts may have been made at other times or at the time of the successful act. Illustrative of the latter and indicative of suicide are the "hesitation" cuts in suicide by cutting, a "snapped" but unfired cartridge ahead of the exploded one, multiple wounds in several methods of suicide (cutting, shooting, etc.), the use of several poisons or a dose many times both the medicinal and lethal dose, etc. So also the suicide may resort to several different methods at the same time.

[53] "His allegedly cheerful spirits up to the time he was seen upon the bridge does not necessarily outweigh the evidence of suicide. It is a matter almost of common knowledge that many who commit suicide do not give any indication beforehand of their self-destructive intent. In many cases suicide results from a sudden impulse. What motivates persons to commit suicide is often a mystery." Waldron v. Met. Life Ins. Co., 347 Pa. 257, 31 A. (2d) 902 (1943). "The physician should not be misled by the previous normal behavior of the individual or the statement by relatives that the decreased was not the person to do such a thing, and that they are quite satisfied that he would never make such an attempt on his life. The most unlikely people sometimes take their own lives and their behavior immediately before the act frequently gives no indication of their intentions. . . . A suicide may be the last person suspected of taking his own life and may do the most surprising things to achieve this end." Kerr, p. 88 et seq.

[54] After a study of 1000 consecutive cases of attempted suicide admitted to Brixton prison in England, Dr. W. Norwood East found that the major causes and motives were: Alcoholic impulse with amnesia (141), alcoholic impulse—memory retained (171), post-alcoholic depression (31), out of work (112), business worries (27), destitution (64), domestic troubles (120), fear of imprisonment or of arrest (41), depression from various causes (20), morbid mental states (18), weak-mindedness (46), neurasthenia (8), epilepsy (10), insanity (123), ulterior purposes (61), other causes, such as shame, mistake under alcohol, etc. (7). See f. n. 15, supra. From our study of cases coming before coroners and courts for decision, there is seldom a single cause or motive for suicide. Usually there is an accumulation of motives culminating in an act of suicide either on impulse or for some slight additional reason which the average, normal man could overcome.

[55] "Preparation for an act is evidence of intent to carry it out. . . . The gun was assembled, loaded, and discharged. No occasion for assembling it existed other than the use to which it was put." Justice Fairchild, dissenting in Tully v. Prud. Ins. Co., 234 Wis. 549, 291 N. W. 804 (1940).

Written or Oral Declarations of the Deceased. Many suicides leave notes removing all doubt concerning the intentional character of the act. Where such a note is discovered, the court should declare as a matter of law that the act was suicidal no matter how accidental the act may seem, unless, of course, the note was ambiguous or was written at some previous time in connection with a contemplated suicide never executed. It may be shown, also, that at some time, not too remote from the time of death, the insured threatened to commit suicide or hinted that he would do so, was tired of life, was going to end it all, would be better off dead, etc.[56] The opposite party is entitled to draw attention to the absence of notes and threats, and the oral and written declarations of the deceased indicating that a short time before his death he made specific plans for the future beyond the time of his death.

2. *Concomitant Evidence*

Physical and Mental States. The likelihood or unlikelihood that the act was intentional may turn upon whether the deceased's mind was clear enough to form the intention, or was so befogged as to make the intention that much easier to execute, or was so befuddled and confused as either to prevent the formation of the intention or increase the probability of an accidental death.[57]

[56] Declarations a long time before the act are inadmissible. Cooley, vol. 6, p. 5469. Statements made to a lawyer are admissible if not privileged. Modern Woodmen of America v. Watkins (C. C. A. 5, Fla.), 132 F. (2d) 352 (1942). As for admissibility of statements made to a physician or in hospital records, the rule will vary from state to state, depending upon statutes relating to privilege, the keeping of hospital records and a determination of what is and what is not hearsay. See Couch, vol. 8, sec. 2201; 75 A. L. R. 378; 120 A. L. R. 1124; N. Y. Life Ins. Co. v. Taylor (C. C. A., Dist. Col.), 147 F. (2d) 297 (1945), and cases cited. Compare Buckminster's Estate v. Comm. Int. Rev. (C. C. A. 2, Tax Court), 147 F. (2d) 331 (1944), disagreeing with the Taylor opinion in its interpretation of the Federal Shop Book Rule, 28 U. S. C. A., Sec. 695, as it applies to hospital records. These appellate courts, it seems, could not agree upon what the United States Supreme Court had ruled in construing the same statute! Palmer v. Hoffman, 318 U. S. 109, 63 S. Ct. 477, 87 L. Ed. 645, 144 A. L. R. 719. In Bolts v. Union Central Life Ins. Co. (City Ct.), 20 N. Y. S. (2d) 675 (1940), the court held that the deceased's statement to a doctor that she did not care to live was not privileged since the doctor already knew that she had attempted her life and the statement was unnecessary to the treatment. The court laid down the generally recognized rule that a physician is barred from testifying to any fact elicited which is necessary to his diagnosis or treatment or may tend to disgrace the memory of the patient or is confidential.

[57] Although most policies contain a clause excluding death from suicide, sane or insane, it has been held in some states that since suicide involves an intentional act, the insured must have had the mind and mentality enough to know that he was taking his own life. 153 A. L. R. 801. A self-inflicted injury may be accidental when received in a delirium or during intoxication or while in some other mental condition where suicide was not intended. Therefore, it is important to determine the deceased's state of mind when the act was committed. Appleman, vol. 1, p. 438, and examples there cited. The use of drugs may so confuse the mind as to preclude any intention of suicide and even cause accidents. Feldmann v. Connecticut Mutual Life Ins. Co. (C. C. A. 8, Mo.), 142 F. (2d) 628 (1944). Compare Aubuchon v. Met. Life Ins. Co. (C. C. A. 8, Mo.), 142 F. (2d) 20 (1944); see also majority and dissenting opinions in Lincoln Petroleum Co. v. N. Y. Life Ins. Co. (C. C. A. 7, Ill.), 115 F. (2d) 73 (1940). In that case the deceased, while intoxicated, quarrelled with his wife, who, to scare him climbed onto the sill of a fourth floor hotel window, then slipped and fell, slumped in a heap but unhurt. He must have thought she was dead, for he edged himself across the sill (which was 3 feet high and in back of a radiator) and then, ignoring warning cries, plunged down head-first with a shout of "Down I come." A jury found in favor of his widow—an accidental death. One judge on appeal thought that the death would be accidental if his death resulted "from an unreasonable or foolish act committed while under the influence of intoxicating liquor or while suffering from great mental or emotional shock." The other two judges reversed the judgment holding: "Whether his mind was cleared by

Was the deceased confused, drugged, intoxicated, insane or in a somnambulistic, "automatic," compulsive, impulsive or delirious state? The same answer may be used to argue both for and against accident! So, also, it may be found that the deceased, at the time of his death, had other physical disabilities or abilities which give a clue to what took place when he died.[58] He may have been epileptic or otherwise afflicted with diseases causing syncope, convulsive or psychomotor actions.[59] He may have been in a physical state such that a medicinal dose of a poison taken without suicidal intention resulted directly or indirectly in death.[60] He may have had a physical illness producing delirium.[61]

the shocking and sobering spectacle he had just witnessed or whether his drunken condition still clogged his mental faculties is immaterial for the exception to insurance liability in each policy covered 'self destruction,' 'whether sane or insane.' Avoidance of this exception is not shown either by the action of one mentally perplexed to the point of insanity or by the less disturbed but nevertheless highly confused mental state of one who is badly intoxicated. A fair construction of this exception clause necessitates a reasonable application to facts. Courts are not justified in holding that it applies to self-destruction by one who is insane, but does not cover self-destruction by one who is drunk, but whose drunken mental state does not reach the point of alcoholic insanity." The ramifications of this question are enormous, but lead us into theories of accidental means and beyond the scope of this paper.

[58] Eye removed at hospital explaining fall from hospital window. Smith v. Durham Life Ins. Co., 202 S. C. 392, 25 S. E. (2d) 247 (1943). Subject to "fainting spells" (not enough, though other evidence of accident was present). Brotherhood of Maintenance of Way Employees v. Page, 197 Ark. 498, 123 S. W. (2d) 536 (1939). Physical impairment of hand accounting for unintended discharge or abnormal manner of handling gun. K. C. Life Ins. Co. v. Bowman (C. C. A. 9, Idaho), 102 F. (2d) 510 (1939); Union Central Life Ins. Co. v. Cooper (C. C. A. 5, Ala.), 115 F. (2d) 222 (1940). Under opiates and ill in bed, death from burning. Brooks v. Met. Life Ins. Co. (Cal. Sup. Ct. in banc) 163 P. (2d) 689 (1945), disagreeing with opposite conclusion of the California Court of Appeals (Cal. A.), 159 P. (2d) 424 (1945). Sleepy and fell off bridge. Hall v. Progressive Life Ins. Co., 61 Ga. A. 792, 7 S. E. (2d) 606 (1940). The length of the deceased's arms may disprove suicide by a shotgun wound. Pythias Knights' Supreme Lodge v. Beck, 181 U. S. 49, 21 S. Ct. 532, 45 L. Ed. 741 (1900) is illustrative of one of many such cases.

[59] Lennox, W. G.: Amnesia, real and feigned, Am. Jr. Psychiat., 1943, xcix, 732; Smith, Hubert W.: Scientific proof and relations of law and medicine, Clinics, 1943, i, 1353.

[60] A curious but too frequent result of imperfect reporting in medical literature is illustrated by the article of Robert Richards, a lecturer in Forensic Medicine at the University of Aberdeen in Scotland, published in 1934 in the British Med. Jr., i, 331. He proposed the theory of "automatism" to explain three cases in which patients later claimed that they emptied a bottle of barbiturates without remembering having done so. This theory, derived from inadequate data and without questioning the motives of these patients who may have been concealing an unsuccessful suicide, was eagerly picked up by other writers anxious to condemn barbiturates and soon became a medical fact, though not a single other such case has been reported since! Enough writers repeated the generalization of Richards to place the theory, unquestioned, in several good books on pharmacology. (How frequently has this happened in medicine?) Ten years later it became the plaintiff's theory in a suit for accidental death benefits. Feldmann v. Conn. Mut. Life Ins. Co. (C. C. A. 8, Mo.), 142 F. (2d) 628 (1944). On the first trial the jury found that the death was accidental, not suicidal, but the result of a "poison," an excepted risk. Judgment for the defendant was reversed because the trial judge failed to define "poison." A retrial resulted in a finding that the insured died of heart disease—not accident, suicide or poison, and the beneficiary did not appeal. The barbiturates are fast becoming a popular method of suicide, although, fortunately, many attempts are unsuccessful because the victim usually falls into a deep coma which may last several days before death ensues, and during this time picrotoxin and other drugs stimulating the higher centers may be administered. It would be unfortunate if the unfounded assumptions of Richards, who borrowed the favorite defense of "automatism" from criminal lawyers to excuse attempted suicides by barbiturates, should gain further currency in legal medicine.

[61] Christensen v. New England Mutual Life Ins. Co., 71 Ga. A. 393, 31 S. E. (2d) 214 (1944), s. c., 197 Ga. 807, 30 S. E. (2d) 471, s. c. (unreported, Ga. A.), 9 CCH Life Cases 268.

Occasion to Use the Lethal Agency. The jury will want to know whether the insured was on a hunting trip or cleaning his gun at the time of its discharge. Did he even intend to go hunting and could that be why he happened to be dragging it from the closet at the time it accidentally went off in his *mouth?*[62] Was the deceased addicted to laudanum, an overdose of which killed him, and did he think that he was drinking wine?[63] Why was the gas stove unlit—had the deceased been using it to cook? Where was he going and for what reason was he driving back and forth across the railroad track,[64] or parked nearby until just before the train arrived,[65] or sitting on the rail?[66]

The Time and Place of the Act. Most suicides are committed in secluded places, in the basement, attic, bathroom, lying in bed, behind locked doors, in the garage or woods, etc.[67] A time and place is chosen when the attempt will not be arrested, and so that the deed will not be uncovered until after resuscitation would be useless. By carefully tracing the movement of the deceased, one may deduce the plan and intention which led up to the act. On the other hand, a similar tracking of his actions may point to the fact that the deceased desired, intended and definitely planned to live beyond the fatal moment of an accidental death.

3. Subsequent Evidence

Conduct of the Deceased. If the deceased accidentally swallowed lysol, he surely would have cried out and sought help because his mistake would have been known immediately.[68] Sometimes the mortally wounded suicide will readily admit what he has done. Others refuse to talk. Still others may claim that it was accidental. Any statement or refusal to talk or indifference to the then known harm, or effort to prevent resuscitation may be proved to show that the act was either suicidal or unintentional.[69]

Conduct and Statements of Others. Relatives of the deceased will often disclose methods, motives, previous attempts and other valuable evidence

[62] This was the famous case of N. Y. Life Ins. Co. v. Gamer (C. C. A. 9, Mont.), 76 F. (2d) 543 (1935), s. c. (C. C. A. 9, Mont.), 90 F. (2d) 817 (1937), s. c. 303 U. S. 161, 38 S. Ct. 500, 82 L. Ed. 726 (1938), s. c. (C. C. A. 9, Mont.), 106 F. (2d) 375 (1939), s. c. 308 U. S. 621, 60 S. Ct. 294, 84 L. Ed. 518 (1939).

[63] Ingersoll v. Knights of Goldeh Rule (C. C., Ga.), 47 Fed. 272 (1891).

[64] Aetna Life Ins. Co. v. Newbern (C. C. A. 8, Ark.), 127 F. (2d) 171 (1942).

[65] Aydelotte v. Met. Life Ins. Co., 124 N. J. L. 266, 11 A. (2d) 122 (1940).

[66] Dixon v. Met. Life Ins. Co., 136 Pa. S. 573, 7 A. (2d) 549 (1939).

[67] So also it may be important to consider that the deceased did not bide his time, but was killed before witnesses, indicating, possibly, an accident. Oubre v. Mutual Life Ins. Co. of N. Y. (La. A.), 21 So. (2d) 191 (1945).

[68] Lindblom v. Met. Life Ins. Co., 210 App. Div. 177, 205 N. Y. S. 505 (1924). "The odor, taste and burning quality were sufficient to enable decedent to identify the acid and quickly emit it if accidentally taken, and yet a considerable quantity of it was found in his stomach." Carroll v. Prud. Ins. Co., 125 N. J. L. 397, 15 A. (2d) 810 (1940).

[69] Hamilton v. Met. Life Ins. Co., 71 Ga. A. 784, 32 S. E. (2d) 540 (1944). On the other hand, the deceased may have sought assistance and then have been cooperative and hopeful of recovery, indicating accident. Union Central Life Ins. Co. v. Cooper (C. C. A. 5, Ala.), 115 F. (2d) 222 (1940); Walker v. Prud. Ins. Co. (C. C. A. 5, Fla.), 127 F. (2d) 938 (1942).

shortly after a suicide and before the advantages of silence are fully known. Any statement of an interested party to a subsequent claim may be admitted in evidence as an admission against interest. Statements to police, newspaper reporters, friends, the undertaker and the coroner may be checked for this type of evidence.[70] Proofs submitted to an insurance company signed by the beneficiary or by an attending physician or containing a copy of the coroner's verdict are admissible as the beneficiary's admissions against interest.[71] They are not admissible when offered in evidence by the beneficiary because they are hearsay and self-serving declarations. Sometimes the interested party may attempt to conceal evidence or will refrain from producing it, though available to such party alone. In such case a legitimate inference of fact (evidence) arises that the evidence would be unfavorable to that party.

Death Certificates and Coroners' Verdicts. We have mentioned that in many states the death certificate is prima facie evidence of the *facts* therein contained.[72] This may or may not permit the introduction in evidence of the death certificate to show the signer's *belief* that the death was "probably" accidental or suicidal, or, if suicidal, "due to a temporary mental aberration."

Almost all states have ruled that the coroner's verdict is not admissible in evidence because it is ex parte and hearsay. An exception exists where the inquest verdict is voluntarily made a part of the proofs submitted to the company and was not required by it. As previously shown, the verdict in such a case is admissible against the beneficiary, but not in his or her favor, because it was made a part of her own proofs to the company.

V

SPECIFIC PROBLEMS RELATED TO VARIOUS VIOLENT DEATHS

The choice of method and the rate of suicide vary widely according to age, sex, race, nationality, rural or urban nature of the population, geographical area, and several other factors. There also seems to be some discrepancy between the figures reported by various compilers of statistics. No single case can be determined by statistical probabilities or the "average" case or what "usually" happens, because in the whole field of this problem there is

[70] A doctor or other witness, expert or lay, cannot testify that in his opinion the death was accidental or suicidal because that would invade the province of the jury on an ultimate issue. Cooley, vol. 6, p. 5472; N. Y. Life Ins. Co. v. Ittner, 62 Ga. A. 31, 8 S. E. (2d) 582 (1940), ruling, however, that any witness in a position to know may testify that in his opinion the wound could or could not have been self-inflicted; Furbush v. Maryland Casualty Co., 131 Mich. 234, 91 N. W. 135 (1902), opinion that deceased was murdered.

[71] Gordon v. Mutual Life Ins. Co. of N. Y. (D. C., La.) 37 F. Supp. 873 (1941); Cooley, vol. 6, pp. 5466, 5477. Proofs are not conclusive if explained as made under great stress and emotion, without reading them, under misapprehension of the facts or in ignorance of material matters subsequently ascertained. Ibid. Proofs made to other companies are also admissible. This source of information is frequently lucrative, but is often neglected by investigators. Fleetwood v. Pacific Mutual Life Insurance Co., 246 Ala. 571, 21 So. (2d) 696 (1945).

[72] 17 A. L. R. 359; 42 A. L. R. 1454; 96 A. L. R. 324; Couch, vol. 8, p. 7232; Cooley, vol. 6, p. 5466 ff.

almost no general rule without its exceptions,[73] and, more important, the court will not admit this type of evidence since the court is concerned only with the case on trial.

It is the almost unanimous opinion of medical jurists, with whom we concur, that except in the case of suicide by firearms the medical jurist is of little practical assistance in determining whether any one death was accidental or suicidal.[74] In other words, in over two-thirds of all cases of violent death where the ultimate decision is suicide, that opinion or finding must depend almost entirely upon other evidence than that of the medical witness, with very few exceptions, and *assuming, of course, that the physical cause of death has been established.* That assumption we carry forward into and throughout the following discussion of evidentiary aids in distinguishing between accident and suicide, according to the cause of the violent death.

A

Gunshot Wounds

1. What Was the Occasion for Use of the Gun?

This topic as a general subject of inquiry in all cases was discussed in IV, B, 2, supra. It may be important to determine whether the deceased

[73] "The method of his exit from life was certainly unusual, but the instances are legion of one determined to die seeking strange or bizarre methods of encompassing death." Home Life Ins. Co. v. Moon (C. C. A. 4, W. Va.) 110 F. (2d) 184 (1940).

[74] If this paper has any value at all it will be in verifying this statement which appears in authoritative sources in general form and in specific reference to various types of violent deaths. We cite here the general statements and will footnote more specific conclusions as each method of suicide is examined. Webster terms the medical evidence as "absolutely necessary" (p. 157) in determining the answer to the question of accident, suicide, or homicide, but cautions that "many of the circumstances are such that the medical witness must know them before he is in a proper position to bear just witness as to the facts" (p. 151). Glaister regards the "whole general question of wounding with respect to accidental suicidal or homicidal causation . . . a field . . . too wide . . . to permit of the statement of such guiding principles as might be of absolute value to the student" (p. 381) ". . . all that can be said is, that the whole circumstances of the wounding, and the environment of the body when found, must be completely observed, considered, and weighed, before a pronouncement of opinion is made . . ." (p. 389). Gonzales states that "accidental deaths can be diagnosed only from the circumstances of the case combined with the results obtained at autopsy" (p. 5) but notes that "it is not possible in all cases to say whether death is homicidal, suicidal or accidental" (p. 108). According to Smith, "the final question . . . can be decided only after a careful consideration of the whole of the facts as well as of any statements made by witnesses" (p. 124). Kerr considers the evidence relating to the circumstances of the death "just as important as the actual violence found on the body" (p. 93). Ewell concluded that "whether [wounds] are suicidal, accidental or homicidal is frequently impossible to determine; and when it can be determined, must depend upon the application of the ordinary rules of evidence and not upon the medical expert." Ewell, M. D.: A Manual of Medical Jurisprudence, ed. 2, 1909, Little, Brown & Co., Boston. See also: Herzog, pp. 280, 286.

Among the writers of the last century Taylor commented that "we cannot always obtain certainty in a question of this kind—the facts will often allow us to speak only with different degrees of probability" (p. 266). "Circumstantial evidence is commonly sufficient to show whether a wound has been received accidentally or not; but as an accidental wound may sometimes resemble one of homicidal or suicidal origin, so it follows that it is not always possible for a medical jurist to decide the question peremptorily from a mere inspection of the wound" (p. 270). See also: Wharton & Stillé, vol. 2, p. 672; Witthaus & Becker, vol. 2, p. 72, 94.

was hunting or even said that he intended to go hunting, or usually carried a gun with him, or was cleaning or repairing or wrapping the gun, or said that he intended to do so.[75] The number of shells exploded and unexploded, in the gun or nearby may indicate that the deceased was loading or unloading the gun or thought that the gun was unloaded, or on the other hand, had deliberately loaded the gun for self-destruction. If he was twirling the gun [76] or "pranking" with it [77] or demonstrating how another had committed suicide [78] or was playing "Russian roulette," [79] then the occasion for the use of the gun would not be for the purpose of suicide, and the inference and conclusion of accident might be drawn. On the other hand, the proponent of the suicide theory will endeavor to show that there was no occasion other than suicide to use the firearm, that no rags or cleaning equipment were near, that no hunting trip was under way or proposed, that the gun was evidently deliberately loaded and discharged, or that the deceased bought, borrowed or assembled the gun for no other apparent purpose.

2. Who or What Discharged the Gun?

In favor of the theory of accident it may be shown that the gun was old, rusty, "tricky" or "easy on the trigger," had no safety device or a defective one, could be (by tests) [80] and had been (from previous experience) dis-

[75] Walker v. Prud. Ins. Co. (C. C. A. 5, Fla.) 127 F. (2d) 938 (1942); Lewis v. N. Y. Life Ins. Co., 113 Mont. 151, 124 P. (2d) 579 (1942); Mut. Life Ins. Co. v. Graves (C. C. A. 3, Pa.) 25 F. (2d) 705 (1928). In one case the insured, former mayor of his town, was under indictment for embezzlement and had far-advanced cancer of the throat and mouth affecting his speech, hearing and sight. One afternoon he locked himself in the bathroom, clad in his pajamas, with an Iver-Johnson revolver which had not been used for years. It could not be discharged except by pulling the trigger. A shot sounded and he later unlocked the door, walked to a bed and collapsed with a contact wound just under his heart. There was no hole in the pajamas. The court held that there was sufficient evidence of accident, because, among other things, he shot himself only once, did not inflict a wound which would immediately kill himself, did not admit that he had attempted to commit suicide, and (of all reasons) there was a dust rag nearby and it was the 4th of July so he might have been cleaning the gun preparatory to celebrating the American Holiday, a practice the court judiciously noticed! Edwards v. Business Men's Assur. Co., 350 Mo. 666, 168 S. W. (2d) 82 (1943). This was the case in which the plaintiff was permitted to submit her case to the jury without electing between the two alternative theories pleaded: (1) that insured accidentally discharged the gun, *or* (2) purposely did so to commit suicide but while insane. An osteopath's testimony that in his opinion the insured's mind was "unsound" was considered substantial evidence of insanity. The jury returned a verdict for the defendant.

[76] N. Y. Life Ins. Co. v. Sparkman (C. C. A. 5, Fla.) 101 F. (2d) 484 (1939).

[77] Met. Life Ins. Co. v. Graves, 201 Ark. 189, 143 S. W. (2d) 1102 (1943).

[78] Aetna Life Ins. Co. v. Kent (C. C. A. 6, Mich.) 73 F. (2d) 685 (1934). In this case a lawyer was showing a friend how he had defended a man charged with murder by contending that the deceased committed suicide. He placed the gun to his head, said it was not loaded, pulled the trigger and killed himself. Accident or suicide? The court found evidence in favor of both theories and affirmed the finding of the jury for the beneficiary.

[79] Pac. Mut. Life Ins. Co. v. Fagan, 292 Ky. 533, 166 S. W. (2d) 1007 (1942).

[80] Brown v. Met. Life Ins. Co., 233 Ia. 5, 7 N. W. (2d) 21 (1942); Scales v. Prud. Ins. Co. (C. C. A. 5, Fla.) 109 F. (2d) 119 (1940); Love v. N. Y. Life Ins. Co. (C. C. A. 5, Miss.) 64 F. (2d) 829 (1933). In the case of Downing v. Met. Life Ins. Co., 314 Ill. A. 222, 41 N. E. (2d) 297 (1941) the court affirmed a verdict for plaintiff holding that tests at the scene near a fence to determine whether a shotgun could have been purposely discharged by the deceased were admissible. However, the court said: "It must be emphasized that evidence of tests or experiments should be received with caution by the trial judge and admitted only where it is certain that they were conducted under circumstances very similar

charged in a variety of ways without pulling the trigger (as by dropping, striking the butt on the floor, striking the hammer or breech, catching the trigger, etc.). The type and make of the firearm are important, it being well known that some guns are much easier to fire accidentally than others.[81] Scratches or marks on surrounding objects may indicate that the gun scraped or struck the object, and the gun itself may have an imprint favoring this theory. The presence of a large number of sticks nearby a man killed by a long-barreled shotgun would destroy part of the inference that he used any one particular stick to push the trigger. So also if his shoes were too large to push the trigger with the toe and if the barrel of the gun was too long for him to reach the trigger, an inference of accident may well arise, there being no pencil, stick or other object nearby with which the deceased could have done the deed. In several cases it has been shown that the gun was fired in a closet where it was left and where it might have become caught on clothes or struck against something to fire it. In other cases the death occurred near a fence or bush or where the deceased or the gun was in position making it awkward for him to handle it and increasing the possibility of its accidental discharge. It may be shown that the gun customarily left "powder burns" on the hand and that there were none on that of the deceased, or that there was a bruise on the hand indicating that he had struck it against something while holding the weapon. In several cases inferences of accident were drawn where the gun might have fallen from or been discharged during removal from a glove compartment of a car, or while on or near the seat of a car, or on a shelf where it was customarily kept.

The deceased may also have had some physical impairment or been subject to "fainting fits" and these facts have been considered proper as increasing the possibility of accident.

In favor of the theory of suicide, it has been shown and considered as proper evidence upon which, with other facts, to raise an inference of self-destruction that the gun was in good condition, required a heavy pull on the trigger, had a safety device or well-guarded trigger, could not be (by tests) and had never been (from experience) accidentally discharged, required grasping of the handle to release a safety, or required a cock and pull of the trigger to fire it. A search of the scene may disclose, generally, no signs of

to those connected with the act to be illustrated thereby." In another case the coroner conducted tests to duplicate "powder burns" found on the deceased and then testified that the gun was at least 13 inches away from the body when discharged. The evidence was admitted over defendant's protest that there was a difference in the cartridges used in the tests and that marks left on paper are different from those on flesh. Lewis v. N. Y. Life Ins. Co., 113 Mont. 151, 124 P. (2d) 579 (1942). Both objections would seem to be valid, except that most authorities on powder marks illustrate their contentions by tests on paper and other inanimate material. Such tests will fairly well duplicate the presence and diameter of powder marks, tattooing, etc. and may be valid, but for that purpose only. The subject requires a more elaborate study than any we have yet found. See: 8 A. L. R. 18; 85 A. L. R. 47. A good example of the impropriety of tests is illustrated in the Gamer case, f. n. 62, supra.

[81] Hatcher, p. 211 et seq. It is possible to remove the magazine of an automatic revolver and forget that a bullet is left in the chamber. See testimony in McLane v. Reliance Life Ins. Co., 192 S. C. 245, 6 S. E. (2d) 13 (1939).

a scuffle or accident or tripping or striking or dropping of the firearm. A recoil mark of a shotgun butt on the ground or floor may point to suicide. If the shotgun barrel was short enough to permit the deceased to reach the trigger, the length of the arms of the deceased should be measured. Many cases come to court without such evidence, and the parties are forced to rely upon such weak evidence as the testimony of a tailor, or that of a relative or friend who tells the jury that the arms of the decedent were "as long as his." The finding of a pencil or stick held in the hand of the deceased is a good sign of suicide where a shotgun has been used, but is not conclusive. The pencil, stick or other object should always be sought and preserved as evidence. The suicide may have occurred in an open place where it could not easily have been caught on anything. A large number of other cases seem to occur while the deceased was lying down on the floor or in bed. The relative positions of the gun, an ejected cartridge, and the body and its extremities may indicate that the deceased either suffered an accident or pulled the trigger himself with intention to end his life, and much evidence of this type is received. It is of questionable value in many cases because of the movements of the body after the shot is fired.

3. Did the Deceased Know of the Danger Imminent?

The fact that the deceased knew how to use and handle firearms and knew the dangers involved may be argued both for and against suicide. Of more importance is a determination of whether he knew that the gun was loaded. In some cases it can be shown that the deceased probably did not know that a shell was in the weapon while in other cases the opposite can be proved by direct or circumstantial evidence.[82]

Where the deceased is found with more than one self-inflicted gunshot wound, or where there are "snapped" but unfired shells next to the exploded cartridge, then it should be strongly inferred that the deceased knew that the gun was loaded, and that he deliberately pulled the trigger with the gun turned upon himself.[83]

[82] Tully v. Prud. Ins. Co., 234 Wis. 549, 291 N. W. 804 (1940). A borderline case is that of Pac. Mut. Life Ins. Co. v. Fagan, 292 Ky. 533, 166 S. W. (2d) 1007 (1942) where deceased "unloaded" a pistol with nine chambers, shortly thereafter demonstrated how the Russians played "roulette" with death and lost the game, falling with a "surprised" look on his face. There were two more unexploded shells found in the gun.

[83] Central States Life Ins. Co. v. McElwee, 199 Ark. 410, 133 S. W. (2d) 881 (1939), where the insured fired five shots, three of them entering his left chest in a three inch circle. The court said: "No reasonable man could conclude that McElwee shot himself accidentally five times at intervals of a minute or more." And see: Domanowski v. Prud. Ins. Co., 116 N. J. L. 247, 182 Atl. 906 (1936); Cruse v. Union Central Life Ins. Co. (D. C., Tex.) 59 F. Supp. 504 (1945). In the last case cited the medical testimony was in conflict whether it was physically possible for a man to shoot himself in or near the heart five or six times. The experts for the plaintiff seemed to ignore the fact that an eyewitness entered the room and found the insured on the floor attempting to draw the hammer back on the gun. He lived for 30 minutes until he reached the hospital. Anyone who reads very far into reliably reported cases of violent wounds will find that the slightest blow may render a person unconscious or kill him, while in other cases unbelievably severe injuries may still be consistent with consciousness, voluntary movement and complete recovery.

4. Where Was the Gun When Fired?

This is an inquiry in which scientific evidence offers substantial aid.

The general considerations that are recommended for all types of medico-legal inquiry into violent death apply in this instance also, since the evidentiary aid of scientific data relating to the use of firearms alone will not determine whether or not a gunshot wound was inflicted intentionally by the deceased or another person.

The following discussion, so far as it relates to scientific determinations of the location of the gun when fired, is, for the most part, drawn from several recent and reliable authorities which may be found in the Additional References to this paper.[84] So far as the reported cases of law are concerned, the witnesses, lay and expert, the lawyers, the judges and the juries seem to have been hopelessly confused. A powder mark of one description will not only receive different interpretations in the same case, but will be taken to have exactly opposite meaning in another case. Descriptions are inaccurate and conflicting. Unqualified witnesses pass themselves off as experts. The result would justify the deriding criticism of Jeremy Bentham in 1827: "Good evidence excluded—bad received! Jargon without end—fiction without shame." This will ever be the result until Scientific Proof whips the lying, ignorant witness, the money changing expert and the pettifogging lawyer from the temple of justice.

(*a*) *Physical Findings*

General Considerations. Since a gun cannot be intentionally fired upon one's self when held with the muzzle over 24 inches away, it becomes necessary to determine the type of a wound which may be caused by the particular firearm and cartridge used when such a gun is held *over* two feet from the body. On the other hand, even where the gun muzzle is found to have been *within* two feet of the body, that fact by no means proves that the deceased either fired the gun, or intentionally did so, or (the ultimate question) pulled the trigger intending to kill himself.

The scientific considerations are in part physical, and in part chemical and photographic. A combination of all three types presents the most conclusive form of scientific evidence and the least likelihood of controversy.[85]

Location of the Entrance Wound. In some cases the absence of holes or powder marks on the garments will indicate, as so often happens, that the deceased drew his clothes aside in order to place the gun against his body. The apparent purpose: to be certain to hit a vital spot. So also the clothes

[84] Walker, pp. 500–519; Moritz, pp. 43–66; Vande Grift pp. 423–430; Hatcher, pp. 200–228; Snyder, pp. 55–123. Other references will be footnoted.

[85] For the means by which Scientific Proof is designed to eliminate error and to secure truth, see Smith, H. W.: Components of Proof in Legal Proceedings, 51 Yale L. J. 537, 1942. One of these is "the use of all appropriate methods of corroboration, with accent on diverse sources and types of evidence." Another is "the eventual grading of all types of evidence according to relative probative value." And another is "the development of usable criteria and safeguards in respect to each type of evidence."

may become ignited or bear evidence of a burn or powder marks at the site of the entrance wound and thus give evidence of a close range of fire.[86]

A renewed word of caution: the mere fact that a wound was self-inflicted does not prove that it was intentionally inflicted for the purpose of *felo-de-se*. And, as noted, the trigger of a gun may even be intentionally pulled while the firearm is aimed at a vital spot of the body and yet the intention to commit suicide may be totally lacking.[87] On the other hand, the mere fact that a self-inflicted gunshot wound is found over or near a vital spot of the body raises a strong suspicion of suicide, increasing to a probability where the gun is found to have been held against or in close proximity to the body.[88] In such cases the path of the bullet or shot is usually straight in and through, and, to achieve that end the suicide in some cases will be found in front of a mirror which he used the better to direct the projectile and so successfully to accomplish his purpose.

One writer states that as many as 62 per cent of all suicidal gunshot wounds are found with the entrance in the mouth,[89] but this percentage is not supported by the experience of others.[90] Such wounds are almost conclusive evidence of suicide, and some courts so hold, but the exceptional case which may always be found may be raised to plague the judge.[91] These observa-

[86] Proctor v. Preferred Acc. Ins. Co. (C. C. A. 6, Ky.) 51 F. (2d) 15 (1931); Knapczyk v. Met. Life Ins. Co., 321 Ill. A. 611, 53 N. E. (2d) 484 (1944). Compare Tabor v. Mut. Life Ins. Co. of N. Y. (C. C. A. 2, W. Va.) 13 F. (2d) 765 (1926). In one case the appellate court examined the pajamas and bathrobe of the deceased and reached its own inexpert conclusion concerning what were powder marks and what was grease, disagreeing with some of the witnesses, and concluding that from what *it* found the *jury* could have concluded that the deceased was shot in the back! Mo. State Life Ins. Co. v. West (C. C. A. 10, Okla.) 67 F. (2d) 468 (1933).

[87] The lawyer demonstrating how another man committed suicide. Aetna Life Ins. Co. v. Kent (C. C. A. 6, Mich.), 73 F. (2d) 685 (1934).

[88] "The nature of the wound itself bars any reasonable hypothesis of accident." Mitchell v. New Eng. Mut. Life Ins. Co. (C. C. A. 4, Va.), 123 F. (2d) 246 (1941). But such a finding is not controlling or conclusive. Scott v. Prud. Ins. Co., 203 Minn. 547, 282 N. W. 467 (1938).

[89] Hatcher, p. 209. This writer also says that 18 per cent of all suicidal gunshot wounds are in the temple.

[90] Snyder, p. 80, says that the majority of such wounds are found in the right temple, with wounds in the mouth next most common. One coroner testified in a case for the beneficiary, whose husband was found with a pistol wound below his heart, that in 3½ years he had investigated 500 suicides. One-half of these died by gunshot wounds and all of those, except one, were in the head! Sutcliffe v. Iowa State Trav. Men's Assoc., 119 Ia. 220, 93 N. W. 90 (1903). Such "statistical" evidence is not admissible because it does not prove the cause of death in the case on trial. The amazing thing is that while courts will stoutly adhere to this rule excluding statistical evidence, they will continue to manufacture their own evidence by "judicial notice" of the most unbelievable and scientifically untrue things. They will also indulge in presumptions to assist a party without evidence which the burden of proof requires him to produce. The criticism of Judge Felton, dissenting in N. Y. Life Ins. Co. v. Ittner, 64 Ga. A. 806 14 S. E. (2d) 203 (1941) will ultimately be justified by historical, statistical, and other scientific proof. He said: "It will be seen, upon consideration, that the presumption against suicide does not owe its existence to facts having evidential value. What most people do or do not do has no bearing whatever on whether one particular individual committed suicide or was killed accidentally. . . . That most people love life too well to destroy it is not a fact *about the deceased* from which the presumption springs."

[91] Gamer v. N. Y. Life Ins. Co., f. n. 62, supra, where an abrasion on the lip was, with other slight evidence, sufficient to permit the jury to guess that the rifle accidentally rammed into the deceased's mouth. It was also pointed out that his artificial upper plate would offer no resistance to such an accident.

tions are but one phase of the general rule that suicides seek to reach a vital organ with as little pain or suffering to themselves as possible, and the corollary: a self-inflicted wound to a vital part is presumptive evidence of suicide.[92]

Size and Shape of the Wound. Entrance wounds may be round, oval, elliptical, lacerated or linear, depending on the position of the bullet at the moment it strikes the target and whether or not the muzzle of the gun is in contact with the target. Large, lacerated entrance wounds are associated generally with contact shots, but in gunshot wounds other than contact shots the entrance wound is usually smaller than the exit. No conclusion is warranted as to the caliber of the bullet merely from the size and shape of the wound. Exit wounds produced by projectiles discharged from firearms are more irregular than entrance wounds and characteristically show lacerations extending beyond the margins of the central defect. Exit wounds will not show any burning, bruising, abrading or deposit of metal, smoke or powder. The presence of foreign particles beneath the skin or within the wound will serve to distinguish large contact wounds with lacerations from exit wounds.

Burning, Bruising, and Abrading of the Entrance Wound. Earlier writers referred to burning of the entrance wound as "the brand." It is occasionally termed "scorching" and sometimes "singeing." The burning will vary in degree and extent depending upon the type of powder charge and the position of the muzzle at the time of firing.

The margins of the skin entered by firearms projectiles are invariably bruised and abraded regardless of the distance from which the projectile is fired. In the event that the muzzle of the weapon is in contact with the skin, a "bruise-pattern" or "stamp mark" of the muzzle, the sight, the ejector slide or the retractor spring rod may be imprinted on the target, depending on individual features of the gun.[93] Metallic particles or lubricant derived from the bullet frequently adhere to the margins of the entrance defect. This is called the "contact ring."

Deposits of Gaseous Combustion Products, Metallic Particles and Powder Residues, etc., Within and About the Entrance Wound. Various

[92] Evidence of the location of the wound takes on added significance when it is considered that suicidal wounds of the head in left-handed persons are found in the left side of the head. Frankel v. N. Y. Life Ins. Co. (C. C. A. 10, Okla.), 51 F. (2d) 933 (1931); Mut. Life Ins. Co. of N. Y. v. Hatton (C. C. A. 8, Ia.), 17 F. (2d) 889 (1927); N. Y. Life Ins. Co. v. Bradshaw (C. C. A. 5, Ga.), 2 F. (2d) 457 (1924). Compare: Jovich v. Benefit Assoc. of Ry. Employees, 211 Ia. 945, 265 N. W. 632 (1936). A wound on the left side of the head in a right-handed person is said to indicate accident or homicide, not suicide. Aetna Life Ins. Co. v. Milward, 118 Ky. 716, 82 S. W. 364 (1904). Compare: Inghram v. Nat'l Union, 103 Ia. 395, 72 N. W. 559 (1897). In Edwards v. Business Men's Assur. Co., 350 Mo. 666, 168 S. W. (2d) 82, the deceased shot himself just below the heart. The court, in holding that there was substantial evidence of accident asked, "If the insured intended to commit suicide because of the motives referred to by respondent, why was a wound inflicted that would not produce immediate death?" To which one might reply: "How does the court know that the insured knew the exact location of his heart?" Few laymen realize the high location of the heart and the fact that one-third of it is to the right of the midline.

[93] See photographic illustrations in Gonzales, pp. 232–233; Snyder, p. 66. This fact is seldom revealed to the jury, because it is not carefully noted or is misinterpreted as a part of powder burns." See: Burkett v. N. Y. Life Ins. Co. (C. C. A. 5, Miss.), 56 F. (2d) 105 (1932); Aetna Life Ins. Co. v. Tooley, (C. C. A. 5, Tex.) 16 F. (2d) 243 (1926).

deposits of smoke, metal and powder about entrance wounds have received numerous terms, such as "tattooing," "smudging," "stippling," "fouling," "smoke halo," "powder marking," and "powder residue pattern." In legal proceedings the general phrase "powder burns" is used indiscriminately by doctors, lawyers, witnesses and the courts to apply to any markings observed, without distinguishing between burning, bruising, and the deposit of foreign material. The terms "tattooing," "stippling," "powder marking" and "powder residue pattern" generally refer to the presence of imbedded grains of powder and particles of metal within the target. "Smudging" and "smoke halo" are names generally applied to the deposit on the target of a fine, black or gray dust or soot containing carbon and metals. Moritz uses the name "fouling" to include both "stippling" by powder and "blackening" by smoke. Hatcher speaks of a "powder brand" in referring to eccentric deposits of powder residue or to dissimilar degrees of burning about the wound. In addition to foreign material derived from the ammunition and gun and deposited upon the target, entrance wounds of skin may also show particles of fabric or hair.

(*b*) *Chemical, Photographic and Microscopic Method*

Ordinary examination under the microscope of foreign materials removed from the surface or subsurface of the wound will serve often to identify characteristic substances such as metal, powder, hair or fabric. Radiographic, spectrographic and microchemical methods are not yet suited to general use, but in the hands of experts the results of such studies are invaluable. The other methods noted provide adequate and useful results for general medicolegal investigations and civil proceedings.

Infra-red Photography. Photographs prepared with infra-red sensitive film and infra-red filters will demonstrate not only the presence of a contact ring but will provide also a permanent record of the distribution of combustion residues about the entrance defect. Blood will not interfere with the results if the proper filter is used.

Macrochemical Tests. Walker has shown that powder residues tested with "C" acid (2-naphthylamine) will give characteristic results highly indicative of the presence of such substances. In the case of fabrics the results can be shown in the form of a permanent imprint upon specially treated ordinary photographic paper. In the instance of skin wounds, Moritz suggests the use of paraffin casts of the entrance wound. Melted paraffin is applied to the wound in combination with layers of gauze. When the hardened paraffin and gauze is removed, most of the material deposited on the skin will adhere to the cast. This in turn may be tested with "C" acid.

(*c*) *Direction of the Tract*

In establishing the direction of the bullet tract autopsy studies should be preceded by and combined with photographic representations showing probes

linking both exit and entrance wounds and protruding from each. In the absence of an exit wound probes must be used with caution since false tracts may be created. The results of determining the direction of the tract are not suitable for establishing all of the circumstances of the shooting but, as Moritz points out, serve a useful purpose in confirming or impeaching the testimony of witnesses. Courts have impliedly in many cases and explicitly in others recognized that where a pistol is discharged away from the body there is less likelihood of a direction through the body perpendicular to the surface of entrance than where the gun is held close to or against the body for the purpose of suicide.[94] So also, if the gun is dropped and accidentally fired, it would be almost impossible to produce a wound in the temple straight through the head from one side to another. Therefore, in reconstructing the events attending the discharge of the gun, the position of the deceased as established by the path of the bullet in the body and after it leaves the body may show that the gun was held and discharged in such a way as to indicate a suicide.

(*d*) *Angle of Fire*

A shot cannot enter the body where the angle of incidence is 5° to 10° or less. The shape of the wound may offer some clue to the angle of fire. Thus, angular shots are associated frequently with linear or ovoid wounds; the near side may show abrasion, the extent of which may be roughly proportional to the angle of incidence. Heavier deposits of gases and powder residue are anticipated generally at the near side of the entrance wound, a finding which can best be reproduced by infra-red photography. Other circumstances, however, may lead to a false impression of an angle shot. Several writers have called attention to the frequent eccentric deposit of powder, smoke and metal about the entrance wound in close-range shots, generally ascribed to the deflection of the gases discharged during the recoil of the gun. Should the weapon consistently produce an eccentric pattern with test shots, a basis is provided for an opinion regarding the position of the gun when fired.

(*e*) *Range of Fire*

The presence of burning, or the deposit of powder, metal or smoke about the bullet wound are general indications of a shot fired at close range.[95] In individual cases consideration must be given to the type of ammunition used, the type of gun, the construction of the gun, the type of target and the pos-

[94] On the other hand the location of the entrance wound and the course of the bullet may indicate that the deceased could have fired the shot intentionally only with difficulty and uncertain aim. Cochran v. Mut. Life Ins. Co. (C. C., Ore.), 79 Fed. 46 (1897). A suicide by holding the gun any considerable distance from the body is said to be an unusual and uncertain way to shoot one's self. Hunt v. Ancient Order of Pyramids, 105 Mo. A. 41, 78 S. W. 649 (1904).

[95] As we have noted, the opinions of courts indicate that, although there is much evidence about "powder burns" introduced in almost every case, there is a complete disagreement among witnesses upon both the presence and interpretation of such marks.

sibility of a discharge of the combustion residue and powder grains at the subsurface of the target, as for example in a perfect contact shot.[96] It is uniformly agreed by authorities that no specific conclusions regarding the range of fire are warranted until test shots have been made using the same gun, the same ammunition and the same type of target as were involved in the case under investigation.

It is possible, however, to make certain general statements regarding the range of fire indicated by the presence of certain features of the entrance wound. *Powder residue.* The deposit of powder residue from the discharge of black powder may occur at ranges of 3 to 6 feet or more, while smokeless powder may project none within a few inches of the target. *Metallic deposits.* Walker finds that molten lead derived from the discharged bullet could be detected in decreasing amounts up to 6 inches from the target but Moritz notes that particles of metal can be carried for a distance of several feet from the target. *Smoke.* Walker states that the products of combustion of smokeless powder generally have a range of 12 to 18 inches, while Snyder places it at 18 to 24 inches. Moritz makes the general statement that targets "less than 12 inches from the muzzle . . . will often be blackened by the smoke of the discharge." *Burning.* The range of fire indicated by burning of the target is said to extend rarely beyond 6 inches and to be present invariably at less than 3 inches (Moritz). It is agreed that with smokeless powder no burning may be observed at ranges of a few inches. According to Walker (following Weiman) "singeing" is observed generally with black powder charges at ranges of 8–12 inches, and sometimes at 20 inches. *Contact shots.* Infra-red photographs are well suited to the demonstration of patterned markings corresponding to features of the particular muzzle in contact with the target at the time of discharge. Observations tending to show extensive disruption of the subsurface of the target and the deposit of smoke, metals and powder within the wound rather than upon the surface serve to support the view that the muzzle of the gun

[96] Yet, many courts, after reviewing conflicting, confused evidence of lay and expert witnesses, probably agree with Judge Fox in McDaniel v. Met. Life Ins. Co., 119 W. Va. 650, 195 S. E. 597 (1938): "The authorities on gunshot wounds do not lay down any inflexible or infallible rule as to the presence of powder burns in any case; whether they appear, and if so, to what extent, depends on the character of the gun used, the kind of powder, the distance from the body, and many other conditions and circumstances." See, also, cases collected in 17 Amer. & Eng. Anno. Cases 36, and Herzog, p. 241. In the case of Gamer v. N. Y. Life Ins. Co. (C. C. A. 9, Mont.) 76 F. (2d) 543 (1935), the court in dealing with a rifle wound in the mouth took judicial notice of ricochet phenomena and finally accounted for the location of a bullet hole in the ceiling. It assumed that the inside of the skull was a smooth surface, that the bullet struck at a slight angle, etc.! In the McDaniel case, supra, the pistol and unfired cartridges were turned over to a constable who later died, "and neither the pistol nor the cartridges could be located and produced at the trial." The bullet was found and turned over to a relative of the deceased but he died and that bit of evidence was not produced! The wound was washed by a physician and he "did not observe any powder marks." No inquest was held but the coroner in another state where the body was buried examined it for powder marks after it had been embalmed and prepared for burial. The plaintiff introduced evidence of tests made to show that "powder burns" would have been present if the insured committed suicide. The court, noting all of this, held that the only way that the jury could have reached a verdict for the plaintiff was by indulging in "conjecture and mere possibilities."

was pressed against the target at the moment of firing.[97] Blood found in the barrel or on the muzzle of a gun is evidence of a contact shot.[98] *Shotgun wounds.* Shotgun wounds will show the features of bullet wounds already described but in addition will be distinguished either by the presence of larger wounds where the gun is discharged within a range of 10 feet or by the characteristic dispersion pattern of the shot at greater ranges. In contact shots extensive disruption of the subsurface and even of entire body cavities is likely to occur. In contact shots of the head the entire skull, brain, and face may be distorted beyond recognition.

B

Poisoning

Suicidal poisoning may result from the inhalation of gases, fumes or vapors, or from the taking of liquid or solid poisons by mouth. A death from carbon monoxide inhalation may or may not be a death from a "poison" or "poisoning" within the meaning of an exclusion in an insurance policy, since death usually results from anemic anoxia.[99]

The medical witness, after once determining that death resulted from asphyxiation or poisoning, will be of little assistance in the determination of whether the death was accidental, suicidal or homicidal. Marks on the

[97] N. Y. Life Ins. Co. v. Newport, 1 Wash. (2d) 511, 96 P. (2d) 449 (1939), shotgun wad in the heart; Gordon v. Mut. Life Ins. Co. (D. C., La.), 37 F. Supp. 873 (1941); Travelers Ins. Co. v. Wilkes (C. C. A. 5, Fla.), 76 F. (2d) 701 (1935); Gorham v. Pac. Mut. Life Ins. Co. (C. C. A. 4, N. C.), 114 F. (2d) 97 (1940). In the Gorham case the autopsy physician concluded that the revolver wound to the head was a contact wound. "This was shown by the absence of branding or burning of the skin surrounding the wound, the presence of unexploded particles of powder in the brain where the bullet was found, and injury to the skull which could only be explained by the explosion within the skull of gases injected at the time of the shot. While there would be an absence of branding in the case of a shot made 12 inches or more from the head, as well as in the case of one made with the muzzle of the pistol pressed against the head, the presence of unexploded particles of powder within the wound and the injury to the skull from exploding gases could only be explained on the theory of a contact shot. The expert witness relied on by plaintiff was not present at the autopsy and had never so much as seen the body of the deceased or examined the wound or the condition within the skull. His testimony was entirely hypothetical and devoid of probative value." One interesting and repeated result of contact bullet wounds to the skull is a suggillation of blood around and beneath the eyes and, in some cases, in other parts of the skin or scalp. In one case where such marks were not explained to the court and jury it was held that they might indicate an assault preceding homicide, an accident in other words. On a second trial, these discolorations were explained on a scientific basis so that the court reversed the case outright, denying recovery despite the testimony of a gun expert that it was impossible for the deceased to have fired the gun himself and to have left no "powder burns," as disclosed by his tests! The two opinions in this case should be "required reading" for everyone interested in the problem under discussion. Bryan v. Aetna Life Ins. Co., 25 Tenn. A. 496, 160 S. W. (2d) 423 (1941), s. c. 174 Tenn. 602, 130 S. W. (2d) 85 (1939).

[98] Reliance Life Ins. Co. v. Burgess (C. C. A. 8, Mo.), 112 F. (2d) 234 (1940).

[99] Much of such evidence was received in the case of Cleaver v. Central States Life Ins. Co., 346 Mo. 548, 142 S. W. (2d) 474 (1940), and the court concluded that the question of whether carbon monoxide is a "poison" was one for the jury to decide. This decision should not be criticized until one has attempted for himself to obtain a satisfactory definition of "poison" from scientists or jurists. See 110 A. L. R. 1276; 131 A. L. R. 1061. When is a drug a "medicine" and when is it a "poison"? Is alcohol a poison? How about the barbiturates which have a lethal dose quantitatively less than numerous well-recognized poisons? Compare: Aubuchon v. Met. Life Ins. Co. (C. C. A. 8, Mo.) 142 F. (2d) 20 (1944) and Feldmann v. Conn. Mut. Life Ins. Co. (C. C. A. 8, Mo.) 142 F. (2d) 628 (1944).

body may indicate either an accidental fall or homicidal violence. Almost all other evidence aiding the jury will be non-medical testimony.

1. Asphyxiation

As far as statistics are reliable, it is found that suicide by carbon monoxide asphyxiation accounts for about 25 per cent of all suicides, and is the choice of method in urban centers.[100] The investigator should carefully note the position of the body with relation to the source of the gas and the presence or absence of methods of increasing its concentration or directing it into the deceased's lungs. The suicide is often found leaning over, upon or near a gas stove, and in one case had arranged a small pillow of cloth to make it more comfortable for him to place his forehead upon the grates. A blanket or shirt or other means may be devised as a hood to increase the concentration. Suicides in autos usually conduct the gas from the exhaust with a hose. The windows and doors of the room or car are found closed, and, in many instances small openings are blocked with rags, carpets or papers. More than one stopcock of a gas range will often be opened, and the absence of burnt matches nearby or the absence of cooking utensils on the stove will indicate that there was no accident. It is possible, of course, for the deceased to have fallen, from disease or accident, then to have struck the gas cock, opening it, and died of gas asphyxiation while unconscious from other causes.[101] It may be possible, too, for the gas cock to have been opened unwittingly or accidentally by the deceased or others and then later, while sleeping, to have been overcome by the gas.

When the deceased is found in a garage dead from the inhalation of gas fumes, inquiry should disclose whether the doors had been shut or locked and for what reason. Was the motor still running, and how much gasoline had been consumed, and still remained? Was the ignition key on? Were there any tools lying around or near the deceased, and was there any grease on him? In other words, was there any indication that he was repairing the car? Occasionally it will be shown that the car needed repair, that deceased said he was going to repair it, etc.

It is possible to commit suicide by the inhalation of motor exhaust in the open without a hood by lying with the face close to the pipe.[102] Because of the lack of expert evidence and scientific proof, the usual circumstantial evidence will fail clearly to demonstrate whether death was accidental or suicidal in most cases where the deceased is found dead from the inhalation of motor exhaust fumes. The smallest scraps of evidence will be used to sup-

[100] Cullen T. J. U.: War-time Prosperity Lowers Suicide Rate, The Spectator 151:8, 1943; Statistical Report of the Chief Medical Examiner of New York City, 1941, pp. 4–5; Report of the Maryland Post-Mortem Examiners Commission, Summary of Deaths, 1942; Annual Report of the Chief Medical Examiner of the County of Essex, N. J., 1942, p. 12; Annual Report on Vital Statistics of Mass., 1942, p. 212.

[101] Herzog, p. 229. An analysis of a blood clot (hematoma) in the tissues for carbon monoxide will disclose whether the injury was received before or after the inhalation of the gas.

[102] Snyder, p. 177, has photographic proof. See f. n. 103, infra.

port a theory of accident or homicide, and the courts seem reluctant to accept as conclusive even the strongest evidence of suicide.[103]

2. Liquid and Solid Poisons

(*a*) In what form and manner was the poison taken into the body?

The investigator, medical and non-medical, should first determine if poison caused death, and, if so, what type it was. He will want to know the form in which it was administered, the container in which it was kept, how much was taken and how much was left.[104] The suicide (like many medical witnesses!) usually does not know the lethal dose of the poison used and will take an excessive amount. Death may occur before all of the poison is absorbed from the stomach. That poison which is left in the stomach could not have caused death, unless it was a corrosive poison, and yet time and again the medical examiner will believe that the cause of death has been proved by an analysis of the stomach contents alone without either a qualitative or quantitative analysis of the tissues. Many qualified pathologists are not competent toxicologists, so that in any case of suspected death from poisoning liberal quantities of all body fluids and tissues should be preserved for expert toxicological examination.[105]

The suicide may dilute a corrosive poison or use other methods to mix and administer it, thus indicating his intent. If he obtained it under pretext, that raises a suspicion of suicide.

[103] For instance, in Allison v. Bankers Life Ins. Co., 230 Ia. 995, 299 N. W. 889 (1941), where deceased was found lying near the exhaust pipe of his car in a secluded woods, with his shirt in a position to suggest that it had been used as a hood, and with the ignition on and hand throttle pulled out, the court said: "If decedent had intended suicide by inhaling monoxide gas, it is unlikely he would have attempted it outdoors." But would the court have declared it suicidal had the death occurred in a garage? This speculation about what the *court* would have done had it been in the deceased's shoes may be found throughout judicial opinions. Thus, another court pointed out that the insured surely did not commit suicide with a rifle in a closet, because "a much more plausible appearance of accident could be simulated somewhere on the proposed fishing trip." Gamer v. New York Life Insurance Co. (C. C. A. 9, Mont.), 76 F. (2d) 543 (1935). So uncertain is the evidence in carbon monoxide deaths that although they are unquestionably among the leading methods of suicide, they are very infrequently contested in court proceedings in suits for insurance. Gunshot wounds, on the other hand, are subject to more scientific analysis, and therefore furnish twice as much litigation as all other methods combined.

[104] One of the best illustrations of the unreliable nature of lay testimony and the difficult problem confronting an appellate court is Bock v. New York Life Insurance Co. (unreported, Tenn.), 1 CCH Life Cases 21 (1938). A youth bought potassium cyanide on a pretext and, after taking some of it in his room, walked past his mother into the yard, fell and was supposed to have struck his head. There was no autopsy. The family doctor disagreed with another doctor as to the appearance of the body, the presence or absence of bruises and blood and the cause of death. The sister claimed that she found the bottle of poison in the yard still wrapped. The druggist who sold the poison weighed it and found that 60 grains were missing. The undertaker, a relative of the deceased, said that when he talked to the druggist, the latter was uncertain whether any of the poison had been removed. The jury returned a verdict for the beneficiary, but the court reversed the judgment.

[105] McNally; Walker, Joseph T.: Scientific evidence in poisoning cases, Clinics, 1943, i, 1520; Report on the Autopsy, an outline prepared by a Conference Group on Pathology of the National Research Council, Jr. Tech. Meth. and Bull. Internat. Assoc. Med. Mus. No. XXIII, pp. 65–70, 1943; Maldeis, Howard, J.: Post mortem examination in cases of suspected poisoning, Am. Jr. Clin. Path., 1943, xiii, 165; Gettler, Alexander O.: The significance of some toxicologic procedures in the medicolegal autopsy, Am. Jr. Clin. Path., 1943, xiii, 169; Jetter, Walter W. and McLean, Regina: Biochemical changes in body fluids after death, Am. Jr. Clin. Path., 1943, xiii, 178.

(*b*) What was the deceased's mental and physical reaction to the poison?

The poison may be fairly easy to distinguish from harmless substances by size, shape, color, container, odor and taste. If the poison was caustic, the deceased must have known what was taken as soon as it touched the lips and tongue. Then why was it swallowed? Why was it not expelled? Why were there no burns on the chin or face? The deceased may have been drunk, drugged, or sleepy, or in some other condition explaining why the poison was taken. However, if the deceased discovered what he took before losing consciousness, then he most certainly would have sought aid or made some outcry of pain, unless suicide had been intended.[106]

In many poisoning cases there will be much testimony and disagreement about the outward appearance of the deceased, the "look on his face," and the position of the limbs, all of which may or may not be considered by a jury as indicating accident or suicide or even death by some other means.

(*c*) Did the deceased know the deadly character of the poison taken and the quantity he took?

Idiosyncrasy [107] to a drug, medicine or poison may cause an "accidental result." So also where the deceased did not know either the proper medicinal dose or the lethal dose, it is conceivable that he took an "overdose" of some "medicine" with lethal or poisonous effect.[108] Alcohol or some other substance or condition of the body may have created a synergistic or enhanced effect of a non-lethal dose of "medicine" so that it became poisonous and resulted in death.[109] In almost every litigated case of death by poisoning some effort will be made to show that the deceased mistook the poison for some medicine of similar odor, taste, color, size, shape or container.

(*d*) What was the occasion for use of the poison?

In some cases there could have been no other occasion for the purchase, concealment and then ingestion of the poison except suicide. However, it may be shown that the poison was purchased and used as a medicine or sedative or to kill rats or to use in the deceased's trade. Poisons are found in almost every household, and, unfortunately, many are poorly marked or their proper use unknown.

C

Hanging

Suicide by hanging is common in males [110] and death by hanging is such strong prima facie evidence of suicide that the mere proof of hanging should

[106] f. n. 68, supra.

[107] See: 152 A. L. R. 1286; Bauder, Reginald, I.: Sulfa Drug Poisoning as an Accident, Proc. Ins. Law Sec., Amer. Bar. Assoc., p. 152 (1944).

[108] Would the death be accidental or caused by "accidental means?" See 111 A. L. R. 1286, and annotations cited.

[109] Walker, J. T., op. cit. supra, f. n. 105, p. 1534; Jetter, W. W. and McLean, R.: Synergistic effect of phenobarbital and ethyl alcohol, Arch. Path., 1943, xxxvi, 112; Dille, J. M., and Ahlquist, R. P.: Synergism of alcohol and sodium pentobarbital, Jr. Pharm. and Exper. Therap., 1937, lxi, 385.

[110] Taylor, p. 427; Glaister, p. 218: Four-fifths of such suicides are males. Taylor, p. 427, and see reports in f. n. 100, supra.

overcome the presumption against suicide, dispense with proof of motive and cast the burden of going forward with the evidence upon the party claiming that death was accidental.[111] Accidental hangings are rare, but may occur as a result of "experiments," usually involving youths.[112] If there has been a fall, accidentally resulting in hanging, attendant bruises and other circumstances may show the absence of suicidal motive or mechanism. The medical witness will not be able to show from a mere examination of the body that the death was accidental rather than suicidal,[113] but he may be able to show that it was homicidal, not suicidal, or that the body was strung up either during life or shortly thereafter to conceal the crime.[114] There are numerous cases reported in which persons have hanged themselves in a sitting or even lying position.[115] If the body is completely suspended, there should be a chair or other object nearby to explain self-suspension, otherwise homicide is indicated.[116]

D

Miscellaneous Causes

Blunt Impact. Of the thousands of deaths that occur each year as a result of motor vehicle and train collisions, there are comparatively few due to suicide,[117] for the two reasons which commonly determine the choice of method: (1) the result is not certain, and (2) pain and disability may precede death. It is difficult to distinguish between suicide and accident in this class of cases, and the medical witness can be of no assistance[118] except in finding disease which may have caused an accident. However, eyewitnesses may be able sufficiently to show that the death was deliberate,[119] and other sources of circumstantial evidence mentioned elsewhere in the paper may make the finding conclusive. It is strange that the law, which permits a finding of willful and wanton misconduct on the part of a motorist, is reluctant to reach a similar result where the intent to destroy is directed against one's self.

[111] Webster v. New York Life Ins. Co., 160 La. 854, 107 So. 599 (1926); Taylor, p. 428; Moritz, p. 164; Smith, p. 256; Webster, p. 95.

[112] Taylor, p. 427; Draper, p. 288; Kerr, p. 137; Gonzales, pp. 261–262; Snyder, p. 141.

[113] Witthaus & Becker, vol. 2, p. 241; Taylor, p. 427; Herzog, p. 224; Draper, p. 288; Gonzales, p. 262.

[114] Smith, p. 257; Moritz, pp. 166–167; Snyder, p. 143.

[115] Snyder, p. 142; Smith, p. 256; Gonzales, p. 261; Taylor, p. 430; Draper, p. 288; Witthaus & Becker, vol. 2, p. 281.

[116] Smith, p. 256.

[117] In the City of New York in 1941, there were 1059 suicides of which only 23 resulted from a jump in front of an auto or train. In the same year there were 607 pedestrian highway and train deaths due to accident. See f. n. 100, supra. Neither juries nor courts are willing to find suicide in this class of cases, and a successful defense on that theory is rare.

[118] Kerr, p. 105; Webster, p. 151, and pp. 157–158; Herzog, p. 286; Gonzales, p. 108; Smith, p. 124 and p. 133; Taylor, p. 266; Snyder, pp. 77–78; Glaister, p. 381 and p. 389; Witthaus & Becker, vol. 2, pp. 94–95; Draper, p. 378.

[119] In International Life Insurance Co. v. Carroll (C. C. A. 6, Tenn.), 17 F. (2d) 42 (1927), a federal judge, hopelessly in debt, involved in speculations which wrecked a bank and, called upon to resign by the bar association pending grand jury investigation, which resulted in indictments, ran his car off the road and straight into a ditch. The court held that there was substantial evidence of an accident.

Fall or Jump. The "fall or jump" verdict of coroners' inquests became common in the depression of the early '30's. There is little medical evidence which can be adduced to distinguish between the accidental fall and the suicidal jump, beyond showing, perhaps, physical and mental disorders or impairments which could account for the injury.[120]

However, much circumstantial evidence may be marshalled to help the jury. It should be shown whether the deceased knew of the imminence of danger, whether the hall was dark when he fell down the elevator shaft, etc. There may or may not have been an occasion for the deceased to be in the window, on the balcony or roof or other place from which he fell. He may have been looking down at his wife who had just fallen from the window,[121] or he may have been trying to get a better view of the harbor,[122] or he may have been just casually sitting in the window waiting to sign over his home, insurance and other property to escape a prosecution for embezzlement.[123]

The condition of the shade, window and screen before and after the fall may show that they were all deliberately raised and not accidentally pushed open. Marks on the ledge may show that the deceased climbed into the window. Measurements and photographs should be made of the place from which deceased fell, the relative location of all objects (such as radiators and chairs) in the room to show whether the deceased could or could not have accidentally fallen or whether he deliberately climbed into the opening. The suicide will often sit on a ledge for quite a time before summoning enough courage to jump. Therefore, there are usually more eyewitnesses to suicidal jumps than to other methods of self-destruction.[124] Moreover, they are commonly accomplished in public places, and in some cases with apparent utter disregard of the safety of others.

Examination of the body by the medical witness will not show anything to differentiate a jump from a fall, unless the height was not great, and in such cases it is of slight importance to show the parts of the body which received the first impact.

There is one type of expert evidence to which resort is frequently made. Measurements of the distance of the vertical fall, the distance of the impact from the place of fall, and the weight of the body may be used in computing whether the deceased either fell or jumped. In one case the deceased who had been sick in a hospital, was depressed and had threatened his life, was found 14 feet from the wall of the hospital under his window which was

[120] A discouraging example of poor reasoning is that of Smith v. Durham Life Insurance Co., 202 S. C. 392, 25 S. E. (2d) 247 (1943). The deceased fell or jumped from the fifth-floor window of a hospital. The back of his head was "bashed in" by the fall. This was part of the evidence from which it was inferred that he fell and did not jump!

[121] The unusual, stranger-than-fiction case of Lincoln Pet. Co. v. New York Life Ins. Co. (C. C. A. 7, Ill.), 115 F. (2d) 73 (1940), partially reviewed in f. n. 57, supra.

[122] Connecticut General Life Insurance Co. v. Maher (C. C. A. 9, Colo.), 70 F. (2d) 441 (1934).

[123] Oubre v. Mutual Life Insurance Co. of N. Y. (La. A.) 21 So. (2d) 191 (1945).

[124] Even an eyewitness will not be able, in some cases, to make the distinction between accident and suicide. Compare majority and dissenting opinions in Lincoln Pet. Co. v. New York Life Insurance Co. (C. C. A. 7, Ill.), 115 F. (2d) 73 (1940).

30 feet from the ground. A professor of mathematics testified that the body must have been travelling at about 30 miles an hour (about 45 feet a second) when it hit the ground; that under such circumstances it would not bounce; and that considering the measurements given the position of the body indicated an expenditure of horizontal energy at the window level which would be equal to a 4 foot 4 inch standing broadjump.[125] In another case a physicist computed the horizontal velocity of the body at the window level to be 11 feet per second, equivalent to a trotting run, and said that if the deceased had fallen, rather than jumped from the window 40 feet from the ground, he would have been found only 2 or 3 feet from the wall instead of the actual distance of 17 feet.[126]

Drowning. It is usually impossible for the medical witness to determine whether a death by drowning is accidental, suicidal or homicidal in absence of marks on the body indicating injury before immersion in water.[127] It is often difficult to be certain that death resulted from drowning, especially where the body is recovered days later and has undergone putrefaction or has been damaged by marine life or ship propellors in such a way as to confuse the determination of the cause of such changes and wounds.[128] The medical examiner will be called upon to state how long the body was in the water, whether death occurred before or after immersion, whether certain marks or physical changes occurred before or after entry into water. The answers to these questions may or may not help in determining the circumstances attending the immersion and death. Here again disease, alcoholism and other facts which may be disclosed to the medical examiner or by investigation will assist in determining the cause of drowning.[129]

Did the deceased know of the dangers involved, the depth of the water, the swiftness of the current, the presence of tidal currents or the whirlpool, etc.? What occasion did he have to be near or in the water? Was he on a fishing trip or gathering driftwood? Could he swim? Was the bank steep and the ledge slippery? Was the breath apparently knocked out of him when he became sleepy and fell off the bridge as he was sitting on the rail, and did he struggle with death for a moment before he was pulled under? [130]

In a recent case the deceased was shown to have been in a desperate financial condition, and his relations with his wife were strained. After gambling all night he returned home at 5:00 a.m. His son unchained the door, but neither his son nor wife would speak to him, so he left the house. That afternoon he was found face down, drowned in a lily pond or fish pool

[125] Hill v. New York Life Insurance Co., 322 Ill. A. 690, 54 N. E. (2d) 88 (1944), s. c. 307 Ill. A. 381 N. E. (2d) 183 (1940).

[126] Christensen v. New England Mutual Life Insurance Co., 71 Ga. A. 393, 31 S. E. (2d) 214 (1944), s. c. 197 Ga. A. 807, 30 S. E. (2d) 471 (1943), s. c. (unreported, Ga. A.), 9 CCH Life Cases 268.

[127] Witthaus & Becker, vol. 2, p. 329; Draper, pp. 268–269; Taylor, p. 414; Smith, p. 274; Gonzales, p. 286; Webster, pp. 101–102; Glaister, p. 194; Kerr, p. 130.

[128] Moritz, p. 170; Draper, pp. 261 and 270; Gonzales, pp. 282–286; Witthaus & Becker, vol. 2, pp. 326–332; Smith, p. 269; Taylor, pp. 409–410; Snyder, p. 156.

[129] Gonzales, p. 279; Smith, p. 274; Taylor, p. 415; Draper, p. 268; Snyder, p. 157.

[130] Hall v. Progressive Life Ins. Co., 61 Ga. A. 792, 7 S. E. (2d) 606 (1940).

in back of his home. It was 7 feet long, 5 feet wide and 2 feet deep, and, with the body in it, the water was about 16 inches deep. The steps leading to the pool were unstable and loose; an almost empty whiskey bottle was nearby; there were several deep, long cuts on the deceased's head, one of which a doctor said "was of itself sufficient to cause unconsciousness"; and "what appeared to be blood" was in the pool. It was the theory of the wife, beneficiary under policies providing accidental death benefits for which suit was brought, that her husband went to the fish pool to repair a leak in the bottom or to rearrange the cattails about the location of which he had argued with his wife a few days before; that he had been drinking and lost his balance when the step turned, causing him to fall, strike his head, become unconscious and drown. The jury evidently believed this theory and returned a verdict for the widow. The court on appeal reversed the judgment and ordered judgment for the defendant.[131]

Strangulation, Suffocation, Smothering, Choking, and Asphyxia (other than by Hanging, Drowning and Poisoning). It is rare to find either accidental or suicidal deaths by any of these methods.[132] Most strangulations are homicidal. Attendant circumstances, other than those furnished by the medical jurist, will be of greatest importance.[133] Infancy, alcoholism, drugs, imbecility, epilepsy, and various other diseases, are factors in accidental deaths within this group.[134] Cases of the accidental lodgment of food or other articles in the throat, trachea, or lungs, causing death by obstructive asphyxia, are reported from time to time.[135] Strangulation by one's own

[131] We have omitted one important bit of evidence to show how strong circumstantial evidence may be and yet be misleading. A long suicide note was found at 8:30 a.m., showing the motive (domestic trouble) and advising his wife: "You'd better destroy this note, for anything that happens to me must be an accident. Remember that! It'll make a lot of difference in your insurance. That ought to keep you quiet for money means more to you than anything else, and the knowledge of this note would mean that you would collect about $5,000 less insurance. $5,000 ought to keep your mouth shut, but it's up to you." The wife at first wanted to destroy the note unread; her son prevailed upon her to read it; she did and tore it up, being unable to find a match to burn it, and threw it into a garbage sack. Later she turned over the pieces to the coroner. At trial she testified that her husband had written a "scare note" before, but the court found that her testimony was "uncertain and unsatisfactory." The note found was written two days before death and after a quarrel, during which his wife belittled him in front of others, and, according to her testimony, she had followed him "clear out to the street" to get him "out of the mood he was in." Home Life Ins. Co. v. Moon (C. C. A. 4, W. Va.), 110 F. (2d) 184 (1940). Compare: Bertschinger v. New York Life Ins. Co., 166 Ore. 307, 111 P. (2d) 1016 (1941), where a naturopathic physician guilty of one illegal abortion and on parole told his lawyer he would jump in the river before he would go through another case in court like that. Eight days later he killed a girl by the same method and was told it would be a coroner's case. Two days later he was drowned. A verdict for plaintiff was affirmed. Clearer thinking is found in Koycheff v. Mutual Ben. H. & Acc. Assoc., 305 Mich. 660, 9 N. W. (2d) 883 (1943), and Equitable Life Assur. Soc. v. Irelan (C. C. A. 9 Mont.), 123 F. (2d) 462 (1941), both being cases of drowning.

[132] In New York City in 1941, out of a total of 5,555 violent deaths investigated, there were only 63 deaths from asphyxiation other than hanging, drowning or poisoning. Statistical Report of the Chief Medical Examiner of New York City, 1941, p. 14; Smith, p. 261; Draper, p. 306; Witthaus & Becker, vol. 2, p. 241; Taylor, p. 440; Gonzales, p. 276.

[133] Taylor, p. 445, comments that "without circumstantial evidence the best medical opinion in these cases (of strangulation) will often amount to nothing."

[134] Moritz, p. 162; Smith, p. 263; Gonzales, p. 274.

[135] 21 of the 63 deaths resulting from obstructive asphyxiation in New York City in 1941 were from this means. f. n. 132, supra.

hands or by ligature using a tourniquet device is possible, although medical experts may be found with a contrary opinion.[136] There are a few cases reporting death by the voluntary blockage of the air passages with all manner of missiles, with self-destructive intent, but such persons are usually mentally defective. Electrocution may cause death by respiratory failure or ventricular fibrillation. Several suicides by this method have been reported but accidents are much more common.[137]

One case well illustrates the uncertainty of much evidence introduced in all cases where the problem of accident or suicide must be solved. A man earning $18,000 a year but over $1,000,000 in debt but with no other apparent motive for suicide is said to have contracted neuralgia in his head from a heavy fog. He used an electric heating pad and obtained relief. He was found dead in his room with the cord from the pad in a noose around his neck, and the other end of the cord tied to the wires of an incomplete electric fixture. There was some dispute about the position of his body, the tightness of the cord and whether, as he lay against the wall, the cord was taut. Three physicians who performed an autopsy described a typical hanging bruise running around the neck, a small bruise on the side of the neck, and no evidence of an electric burn. One embalmer said that one mark "seemed like a burned place similar to an electric burn," and the other embalmer said the mark "appeared to be a burn and was brown and parched looking." An electrical expert showed how it was "possible" for a current to pass through the wires and into the body and said that fine wires in the knot were beaded "as though an arc had jumped across which would probably have caused a shock." He also said that 120 to 130 volts were "not supposed" to be enough to kill a man, but that there was uncertainty about it; that some persons were more susceptible to shock than others and that certain parts of the body were more sensitive than others.

The beneficiary first advanced the theory of murder, but this the court rejected for the reason that there was no evidence to support it.

The beneficiary then showed that a few weeks before her husband's death a friend of his was found dead from what appeared to be self-strangulation with a shoestring; that her husband had doubted this as possible and on one occasion undertook to demonstrate with his necktie that this was not possible; that her husband was of "an experimental turn of mind" and must have been attempting to demonstrate that his friend had not committed suicide; and that he probably electrocuted himself accidentally. The court said that this was pure conjecture; that "it is inconceivable that deceased would conduct, in solitude, a pointless experiment of so dangerous a nature"; that "reasonable men do not so recklessly trifle with death"; that whether the deceased died of strangulation, electrocution or both, he was killed as he had pre-arranged; and that the lower court had properly directed a verdict

[136] Kerr states that "suicide by this means is out of the question," p. 138. Compare Gonzales, p. 272; Taylor, p. 440; Witthaus & Becker, vol. 2, p. 241; Smith, p. 260.

[137] Snyder, p. 227; Smith, p. 241; Gonzales, p. 296.

for the defendant. It may also be mentioned that eleven months after the death the coroner held an inquest at which the jury found that the death resulted from "external violence or homicide." The record of the inquest was properly excluded from evidence.[138]

Cutting. It is not uncommon that a person will die of accidentally inflicted incised wounds of the neck or wrists, but in virtually all of such cases the surrounding circumstances will clearly point to the cause of the wounds.[139] The difficulty does not lie in reaching a choice between accident and suicide, but rather in determining whether the wound was suicidal or homicidal.

Suicidal wounds of the neck are usually found above the thyroid cartilage on the left side in right-handed persons and on the right side in left-handed persons.[140] They may be either deep or superficial, regular or irregular, but are usually deep, ragged and slanting diagonally with the deepest cut at the beginning of the stroke.[141] Multiple strokes of the instrument may be found in one wound.[142] Most characteristic of all are several superficial cuts at the beginning of the wound, the so-called "hesitation" marks of the suicide.[143]

An interesting case is that in which a man was found bleeding profusely from a deep cut of his right wrist, a long blood-stained knife nearby. The medical examiner and another physician testified that the wound was self-inflicted because of the presence of "hesitation" marks. No other physician testified. Yet the court held that other evidence made the case one for the jury rather than for the court to determine.[144]

Stabbing. Where a stab wound is made with a knife or other hand-wielded weapon in a suicide, it is usually found in or near the region over the heart, although it may rarely appear elsewhere, and is directed from right to left in a right-handed person and from above downward.[145] Several stab wounds in a circumscribed area indicate suicide rather than homicide, and almost completely exclude accident.[146] Since the *felo-de-se* may stab himself

[138] American National Bank v. Continental Cas. Co. (C. C. A. 6, Tenn.), 70 F. (2d) 97 (1934).

[139] Webster, p. 151; Gonzales, p. 210; Glaister, p. 359; Moritz, p. 38; Taylor, pp. 267–269; Snyder, p. 123; Witthaus & Becker, vol. 2, pp. 80 and 94; Kerr, pp. 94–96 and pp. 103–104; Smith, pp. 124, 128–129; Draper, p. 380.

[140] Moritz, p. 38; Taylor, p. 268; Witthaus & Becker, vol. 2, p. 78; Snyder, p. 127; Smith, pp. 128–129; Gonzales, pp. 211–212.

[141] Kerr, pp. 103–104; Moritz, p. 38; Smith, p. 128.

[142] Kerr, pp. 103–104.

[143] Gonzales, p. 211; Moritz, p. 38; Smith, p. 129, Snyder, p. 127.

[144] Facts purposely omitted, and this time showing the importance of circumstantial evidence (compare f. n. 131, supra), are that the deceased, who apparently had no motive for suicide, was in the grinding and cutlery business and was found at his shop where he had been buffing large knives on a rag buffing wheel with a speed of 1400 r.p.m.; that the wheel was wobbly and had a deep cut in it; that there were blood stains on the ceiling above and the floor below the wheel; that the knife found had been partially buffed; that the deceased had made plans to go to a show with his wife later in the ofternoon; that when his wife arrived at the shop he sought her help. One confusing fact, never explained, was why the deceased did not rush away from the machine and seek aid instead, possibly, of lying down by his machine until he was found. Kirschbaum v. Metropolitan Life Insurance Co., 133 N. J. L. 5, 42 A. (2d) 257 (1945).

[145] Gonzales, p. 207; Smith, p. 129; Draper, p. 380; Witthaus & Becker, vol. 2, pp. 73, 75.

[146] Gonzales, p. 208.

more than once through a single opening and since multiple stabbings indicate suicide, the examiner should carefully determine the course and number of all wounds inside the body. Rarely a butcher or cook may run a knife into the abdomen by accident while drawing it toward himself.[147] Falls and other accidents may cause an impaling of the body on some object, or flying splinters or glass may cause fatal stab wounds.[148] In such cases, and in homicide, the pattern—multiple but general—and direction will usually show that there was no suicide. Surrounding circumstances are important.

A man and his wife were in the kitchen and he was wiping the dishes. As he was wiping a paring knife she heard him say, "Here goes." She looked at him and saw blood on his shirt. He laid down the knife, took a few steps and died shortly thereafter. A knife wound two inches deep had penetrated the heart. He had had business reverses, had threatened self-destruction and had been drinking. His wife, the beneficiary of an accident insurance policy, introduced evidence that he was in good health and spirits, in fair financial condition, had no domestic trouble, was temperate and jovial and liked to play with children, was given to mock heroics and "play acting" and frequently moved a knife toward his body, exclaiming, "Here goes," and then turning the knife just before striking his body with his fist, for the purpose of frightening others with this performance which he called the "Dutch act." The court, in affirming a judgment for the beneficiary, held that there was substantial evidence that he accidentally killed himself.[149]

Burning. Suicide by this method is rare.[150] It may be used for homicide or for concealment of a homicide or suicide by other methods.[151] Death by burning is almost always accidental.[152] Disease may be a direct or indirect cause of the fire and thus possibly affect a recovery for accidental death benefits, but the same rule applies to almost all other apparently violent deaths and is beyond the scope of this paper. A careful consideration of all of the attendant circumstances together with such general inquiries suggested elsewhere in this paper will usually disclose whether the death was accidental or suicidal.[153] Of chief importance will be a search for any marks of violence which may have indicated accident, suicide or homicide preceding the death by burning.

In a recent case the deceased was suffering with rectal cancer and was confined to his room under opiates. He had day and night nurses; the night nurse went off duty at 7 a.m. and the day nurse came on duty at 8 a.m. His wife usually cared for him between 7 and 8 a.m. but on the day of his death left at 7 a.m. with the night nurse to whom the insured had made veiled threats of suicide. When the day nurse arrived at 8 a.m. she found the bed-

[147] Ibid.
[148] Gonzales, p. 208; Snyder, p. 126; Smith, p. 128.
[149] Missouri State Life Insurance Co. v. Pater (C. C. A. 7, Ind.), 15 F. (2d) 737 (1926).
[150] Glaister, p. 257; Smith, p. 237; Webster, p. 116; Gonzales, p. 292.
[151] Smith, p. 236; Snyder, p. 170; Gonzales, p. 292.
[152] Glaister, p. 257; Taylor, p. 394; Kerr, p. 109; Smith, p. 236; Webster, p. 116.
[153] Smith, pp. 237–238; Taylor, p. 394; Kerr, p. 109; Webster, p. 116.

room on fire and the doors, which had been unlocked at 7 a.m., locked. The shades, which had been up when the night nurse left, were pulled down. The deceased was found dead with the lower part of his body burned and the odor of inflammable rubbing alcohol on the body, the empty bottle in a basket. There was some dispute whether a gas heater was near to or distant from his bed. It was plaintiff's theory that papers had fallen off the bed and ignited it and that the deceased was unable to help himself in time. A doctor said that with opiates given to the deceased he would not have much feeling in his body. A jury returned a verdict for the defendant, but the lower court granted a new trial on the ground that there was insufficient evidence to support a verdict of suicide. On appeal the conclusion was affirmed by the California Court of appeals. However, the California Supreme Court held that there was sufficient evidence to support a verdict either of accident or suicide but that it had no power to interfere with the trial court's conclusion that the case should be retried.[154]

VI

CONCLUSIONS AND SUMMARY

1. The problem of determining whether a death was accidental or suicidal frequently arises and is of great importance in coroners' investigations, insurance claims and workmen's compensation proceedings. This determination infrequently becomes necessary in several other fields of law.

2. In cases arising in insurance law and under workmen's compensation statutes, procedural rules relating to the burden of proof, presumptions, and the admissibility and sufficiency of evidence substantially affect the final result and in many cases seem as important as real evidence. Strange and unjust decisions of juries and courts have been due to several misconceptions which should no longer be followed: (1) the continued recognition of the presumption against suicide without any effort to reëxamine it from a scientific viewpoint to determine its present-day validity; (2) the application of this presumption in cases where it is obvious that the deceased did not love life or fear death enough to deter him from suicide; (3) the application of this presumption to various types of violent deaths which are statistically proved to be more commonly suicidal than accidental; (4) the adherence to obfuscated rules of evidence and the treatment of the presumption against suicide either as evidence or as a rule of law upon which the court may instruct the jury or as a "fact of life" which the jury may consider along with other evidence in finding that a death was accidental; (5) the failure to distinguish between suits on life policies where the burden of proving suicide is on the defendant, and suits for accidental death benefits where the burden of proving accident is on the plaintiff and no burden of proving suicide is on the insurer; (6) the departure of courts from rules of law universally applied

[154] Brooks v. Metropolitan Life Insurance Co. (Cal.), 163 P. (2d) 689 (1945), s. c. (Cal. A.), 159 P. (2d) 424 (1945).

in all other cases, notably those rules requiring the production of substantial evidence sufficient to remove an issue from the realm of mere guess and speculation.[155]

3. Other factors contributing to unjust results where the accident-or-suicide problem arises are: (1) lack of proper medical, police and other investigation at the scene and time of the injury; (2) failure to pursue all available avenues of investigation indicated at the time; (3) failure to obtain and preserve the evidence in a form in which it may be used without contradiction later on; (4) the free employment of unqualified "experts" to testify about things they do not know but in a way for which their price was paid; (5) incompetence of jurors to decide scientific controversies; (6) prejudice against insurance companies and employers and in favor of injured persons and widows; (7) all of the other unsavory results of ignorance and dishonesty, both in and out of court by lay and expert witnesses.

4. Proof that a death was accidental or suicidal may be made by resort to either direct or circumstantial evidence, and in general this evidence may be described as External or Internal. External Evidence is directed to the physical facts and circumstances surrounding the death from which one may conclude whether the injury was self-inflicted. Internal Evidence is designed to prove whether this self-inflicted injury was intentional, and may be drawn from facts and events preceding, attending or following the injury.

5. Medical jurists have laid down numerous general rules of little assistance in reaching a conclusion in any one particular case in which a death may have been either accidental or suicidal.

6. Specific problems related to various kinds of violent deaths have been examined. Except in the case of gunshot wounds, the medical witness will seldom be able to express an opinion, from an examination of the body alone, whether death was accidental or suicidal. After the physical cause of death is once established, the ultimate decision of whether a death was caused by accident or suicide will depend almost wholly upon non-expert evidence, excepting again a death by gunshot wounds. In this one class of cases, the medical witness with a fundamental knowledge of firearms and reasonably accurate information concerning the weapon and cartridge used can furnish invaluable evidence of the relative position of the gun to the deceased at the time it was fired.

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PENICILLIN IN THE TREATMENT OF PUTRID LUNG ABSCESS *

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During the last two decades our knowledge of the pathogenesis and pathological physiology of putrid lung abscesses has advanced remarkably. This inevitably led to great improvement in the quality of the surgical treatment of this serious disease so that an ever increasing number of such patients are now being treated effectively by surgical means.

At this time it is generally agreed that acute putrid lung abscesses which are accompanied by great toxicity or which show no rapid roentgenological improvement by medical measures, are best treated by early thoracotomy and pneumonostomy. This is also true of abscesses which progress, rupture into the pleural cavity, or give rise to persistent hemoptysis.

In fact there is an increasing number of physicians who believe that all acute putrid lung abscesses, regardless of their size or the severe toxic symptoms they engender, should be treated in this manner as soon as the diagnosis is made. This group has been encouraged in its beliefs by many writers, notably Neuhof and Touroff [1] who in 1940 reported the operative results in 86 consecutive patients with acute putrid lung abscesses, 21 of whom had putrid empyema. Of these, 73 recovered, three died of septic complications post-operatively and four died of diseases other than the lung abscess. However, many other physicians have not been so fortunate in their results with surgical treatment of this serious disease. Sweet [2] in 1940 reported a series of 125 cases of acute putrid lung abscess from the Massachusetts General Hospital. Of those operated on in this group 7.4 per cent died immediately post-operatively, 26.6 per cent died ultimately of the abscess or its complications, and only 43 per cent were cured. Sweet concluded that the results of the surgical treatment of lung abscess were on the whole disappointing. He stated that if there is a good chance of spontaneous recovery operation should be avoided, but if one can be reasonably certain that spontaneous recovery will not occur, then the sooner the operation is performed the better. This attitude echoes an earlier view of Cutler [3] who in 1936 stated that an intensive study of the case histories of the group he presented made it obvious that, if all such cases were submitted to operation, the mortality rate would be considerably cut down, but that in so doing we would submit to operation many patients who would have recovered spontaneously.

As a matter of fact spontaneous recovery occurs only in a small percentage of cases of acute pulmonary abscesses who seek hospital care. In

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From Medical Service of the Harlem Hospital, Dr. Oswald LaRotonda, Medical Director.

the Harlem Hospital in the past few years spontaneous recovery occurred in only eight, or 11 per cent, of 70 such patients. This recovery occurred only in patients who were not very toxic and had small abscesses, usually less than 3 cm. in diameter, with little perifocal pneumonia.

However, it is not the purpose of this discussion to reconcile these opposing views or to inject new material into the controversy, but rather to indicate that because of the views of Sweet, Cutler and others many acute putrid lung abscesses in private practice and in hospitals are observed for protracted periods of time in the hope that they will recover under medical treatment. During this period serious and often fatal complications frequently arise, such as septic embolism, progression of the abscess with the formation of multiple abscesses, fibrosis, atelectasis and bronchiectasis in the lung involved, extension into the opposite lung, and rupture into the pleura with putrid empyema. Moreover, while waiting acute abscesses become chronic abscesses and these rarely if ever recover spontaneously. Most distressing is the fact that patients with chronic abscesses are not cured with simple pneumonostomy, and the mortality rate is high, even with the best surgery, because of many grave complications.

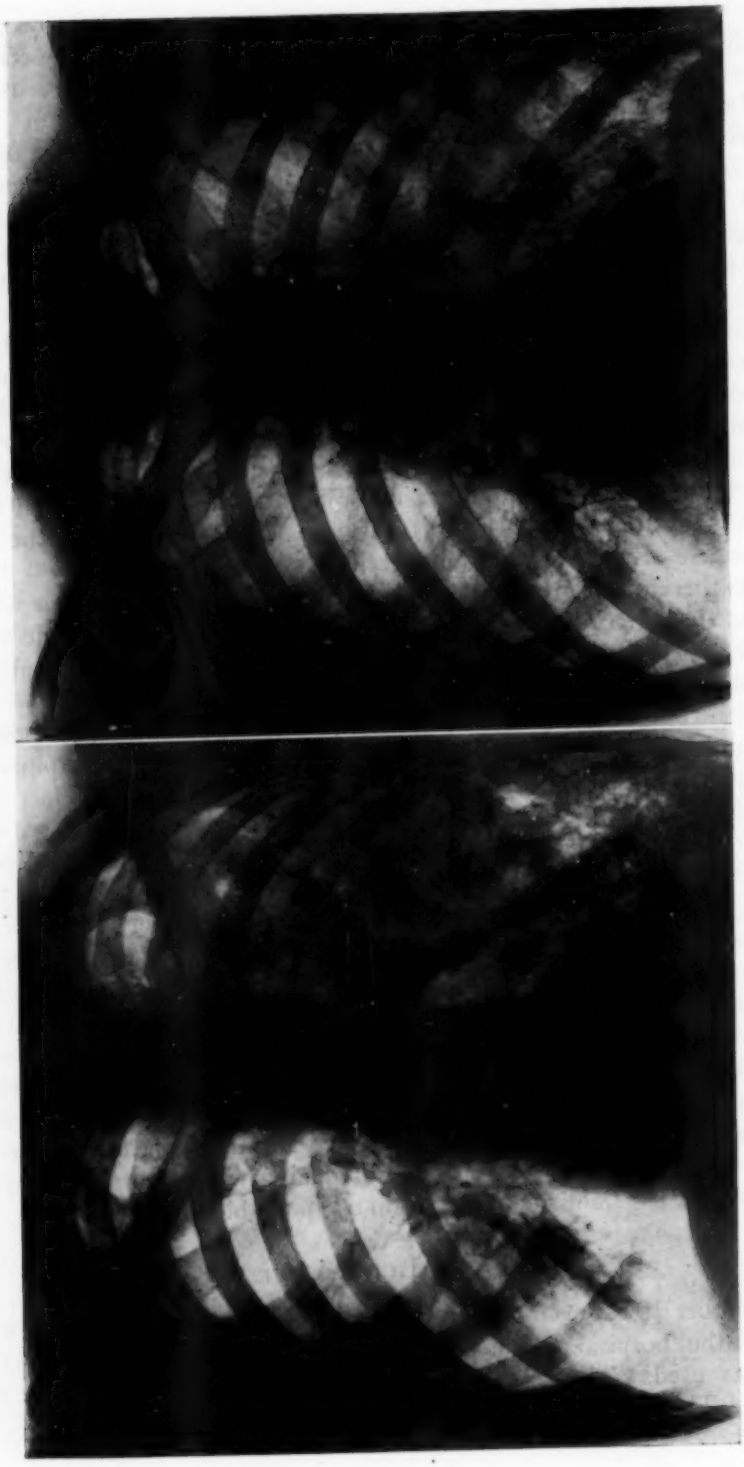
There is therefore a great need for a method of treatment which would (1) increase the rate of recovery without surgical interference, (2) decrease the number and severity of pre- and post-operative complications and (3) make operative treatment generally safer and more effective.

When penicillin became freely available during the past year, we administered it to 13 patients with putrid lung abscess in order to explore its potentialities. Sulfadiazine was employed concurrently despite the fact that, when used by itself, it had no appreciable effect on a small series of patients so treated. It was hoped, however, that it might have a beneficial effect on the perifocal pneumonitis.

Although from 20 to 25 patients with acute putrid lung abscesses are admitted annually to Harlem Hospital, there were only 10 such cases in the hospital during 1944 and six during 1945. We are not certain whether this marked decrease in the last two years was fortuitous or due to the early and frequent use of penicillin in all kinds of pulmonary infection. As soon as the diagnosis of putrid lung abscess was definitely established and confirmed by roentgenograms the patient was given both sulfadiazine and penicillin. The sodium salt of penicillin was administered intramuscularly in doses of 25,000 units every three hours. The sulfadiazine was given in the usual manner, sufficient to establish and maintain adequate blood levels.

Acute Putrid Lung Abscess

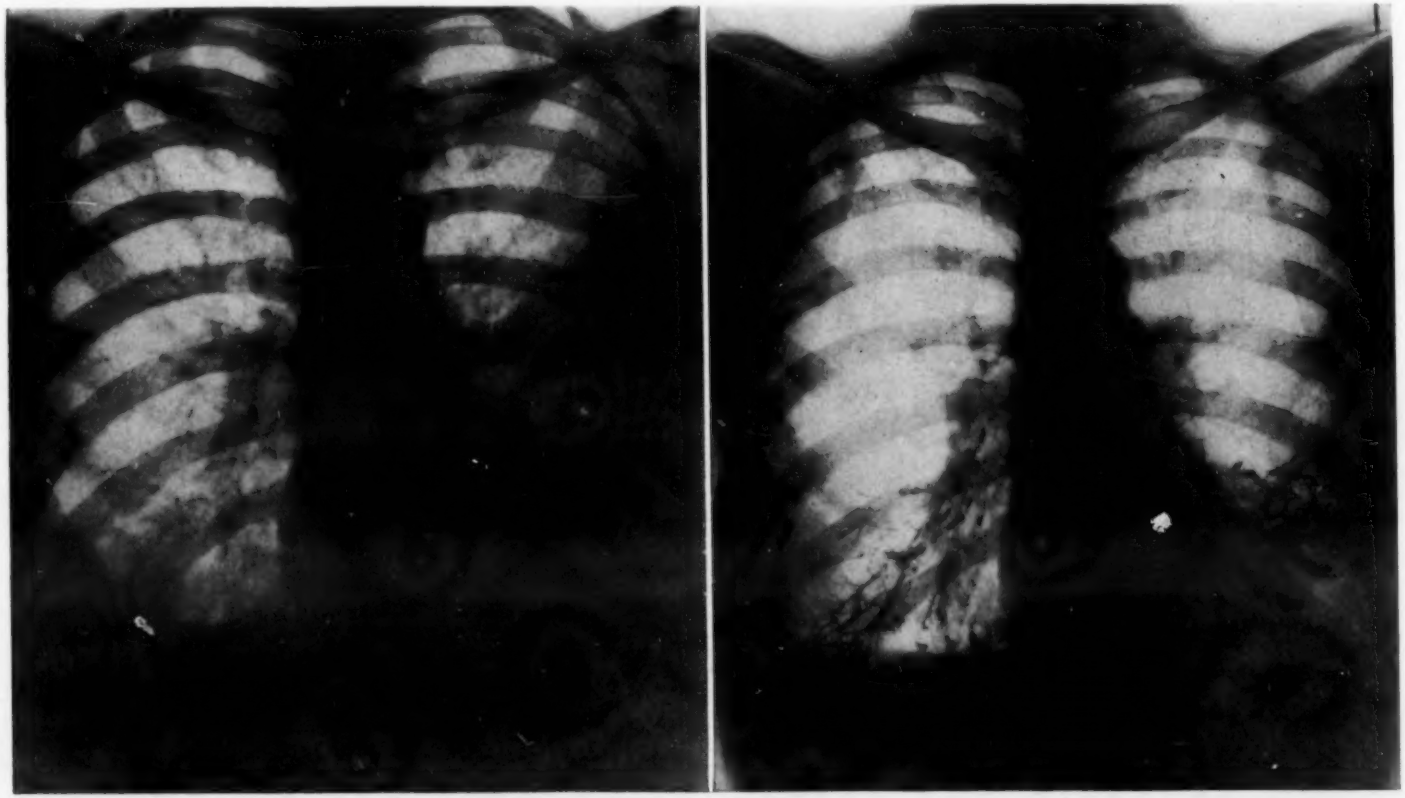
Since October 1944 seven patients with acute putrid lung abscesses were admitted to the hospital. One of these recovered spontaneously before penicillin was used. The remaining six were treated with penicillin and sulfadiazine. These are presented in the order of their admission.



B

A

FIG. 1. *Case 1*, T. J. *A*. Film taken Oct. 24, 1944 showing diffuse infiltration and numerous cavities in the left lower lobe. *B*. Film taken Dec. 5, 1944 showing complete clearing of lesion.



A B

FIG. 2. *Case 2,* I. R. *A.* Film taken April 24, 1945 showing diffuse infiltration with large cavity in the left lower lobe. Consolidation with cavity at right base. *B.* Taken June 21, 1945 shows bronchiectasis in left lower lobe.

Case Reports

Case 1. T. J., a 49 year old male negro was admitted to Harlem Hospital on Oct. 21, 1944. He had suffered from frequent severe attacks of bronchial asthma since early youth. His temperature on admission was 102° F., pulse 120 and respirations 30 per minute. He appeared distressed and seriously ill. He expectorated large quantities of fetid sputum. The thorax was emphysematous in type. Expiratory wheezes were heard throughout both lung fields and scattered moist râles throughout both lower lobes. The admission roentgenogram of the chest revealed confluent consolidation of the entire left lower lobe with the moth-eaten appearance of early diffuse cavitation. A diagnosis of acute putrid lung abscess was made. On Oct. 22, 1944 penicillin and sulfadiazine therapy was started and this was continued to Dec. 5, 1944. He became afebrile and asymptomatic on Oct. 27. Serial chest films revealed continued clearing until Nov. 27 when the chest film was that of a normal lung. He was discharged on Dec. 8, 1944.

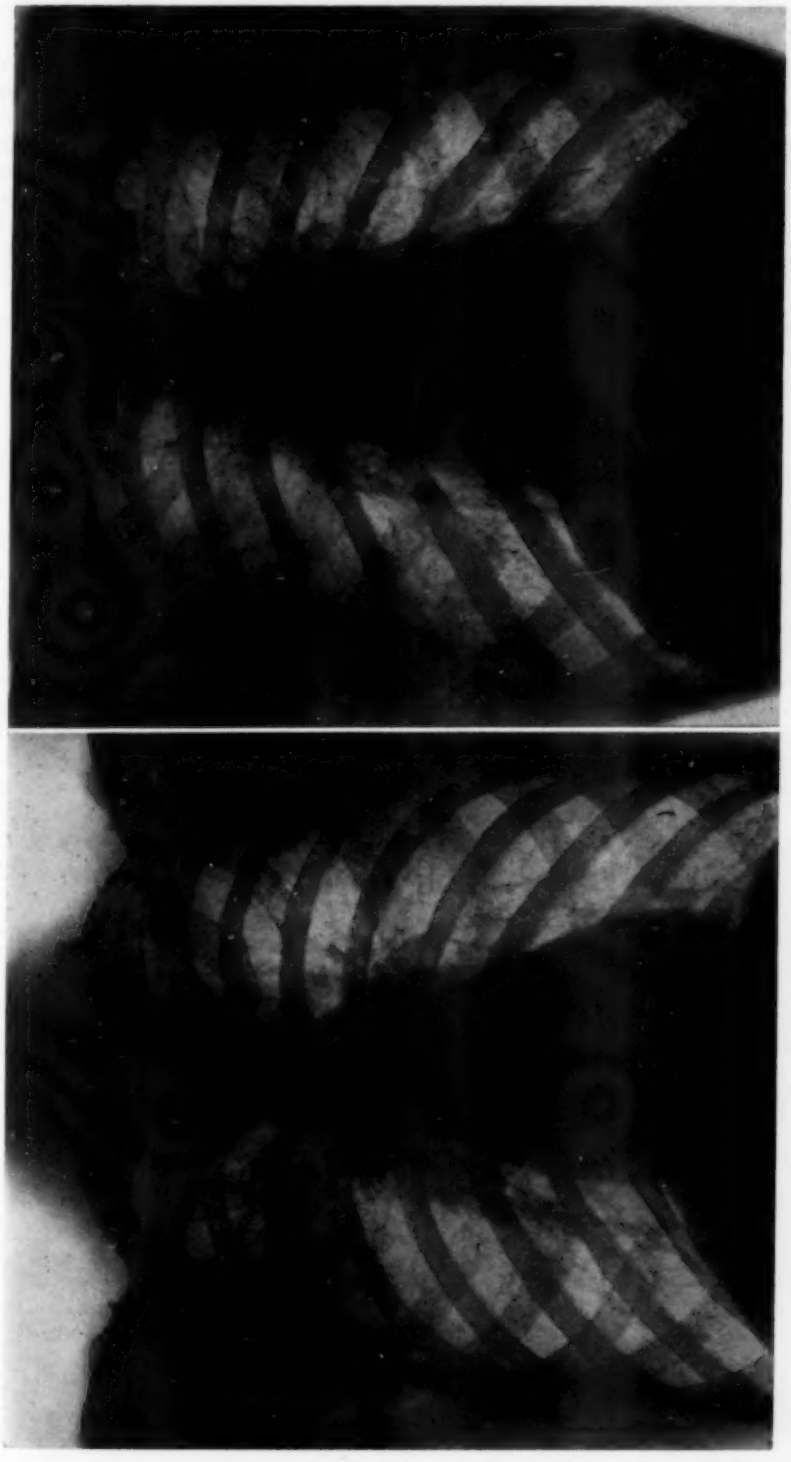
Comment. This patient was seriously ill with bronchial asthma and acute putrid lung abscess. He responded to the penicillin and sulfadiazine therapy with complete disappearance of the lung abscess.

Case 2. I. R., a desperately ill negress of 35 years, was admitted to Harlem Hospital on April 23, 1945. She gave no previous history of illness, operation or injury except cough for the preceding five weeks. Early in April she had a productive cough with blood streaked sputum. On April 16 she noted pain in the right lower chest. This became intense on the twenty-second, when she noted shortness of breath.

On admission her temperature was 103° F., pulse 120 and respirations 32 per minute. The breath was foul and the sputum was profuse and putrid. There were dullness and large moist râles over the lower two-thirds of the left lower lobe. The chest film on admission revealed a large abscess cavity in the left lower lobe with pneumonic involvement of this entire lobe and considerable pneumonia with small abscesses in the lower lobe of the contralateral lung. The sputum contained no acid-fast bacilli. On April 25 a surgical consultation for pneumonostomy was requested and treatment with sulfadiazine and penicillin was started preparatory to the proposed surgical intervention. This treatment was continued until June 15. Three days after the therapy was started she had improved markedly and her temperature fell to 101° F. On May 5 her temperature became normal; she remained asymptomatic and afebrile and obviously was no longer in need of surgical care. Serial roentgenograms revealed continued clearing of the abscess cavities and the pneumonic infiltration. On May 10 the abscesses were no longer visible. On June 21 a bronchogram revealed dilatation of the bronchi of the left lower lobe. She was discharged on June 25, 1945 and has been well and asymptomatic to the present time.

Comment. This patient had bilateral acute putrid lung abscesses with suppurative pneumonia in both lungs. The widespread putrid infection of the lungs cleared fully and promptly on sulfadiazine and penicillin therapy. She was left with a residual bronchiectasis in the left lower lobe. She was gravely ill and it is doubtful that she would have survived the operation which seemed necessary when she was admitted.

Case 3. L. McK., a 38 year old male negro, was admitted to Harlem Hospital on May 5, 1945. Throughout his life he had had frequent sore throats and nasal colds. He was well until April 22, 1945 when he began to cough and expectorate blood streaked sputum. His appetite was poor, he fatigued easily and lost 25 lbs. His



A B

FIG. 3. *Case 3*, L. McK. *A*. Film taken May 14, 1945 showing large abscess with fluid level in upper portion of the right lower lobe. *B*. Film taken July 9, 1945 showing clear lung.

temperature was 104° F., pulse 124 and respirations 22 per minute. He was poorly nourished. The breath and sputum had a foul odor.

Physical examination revealed moist râles over the right infrascapular region. The chest film on admission revealed a huge cavity with a fluid level in the upper portion of the right lower lobe. The sputum contained no acid-fast bacilli. Penicillin and sulfadiazine were started the day after admission and by May 12 his temperature had fallen to 101° F. and the sputum was no longer foul. On May 21 he became asymptomatic and afebrile, and he remained well until his discharge. Serial chest roentgenograms revealed a clearing process in the left lung. The film of June 15 revealed no cavitation and the film of July 9 revealed a normal lung. He was discharged on July 12, 1945.

Comment. This patient had a large acute putrid lung abscess which cleared completely with penicillin and sulfadiazine. There were no pulmonary, pleural or metastatic complications.

Case 4. A. P., a 21 year old Puerto Rican male, was admitted to Harlem Hospital on July 19, 1945. He had been well until three days before admission when he had a chill and became feverish. He complained of pain in the right lower chest associated with nausea and vomiting. He coughed and expectorated foul sputum.

Physical examination revealed dullness and broncho-vesicular breathing over the right lower lobe posteriorly. The temperature was septic in type, fluctuating between 99° and 102° F. The pulse rate was 110 and the respirations 24 per minute. The chest film revealed a cavity with a fluid level in the lower third of the right lung field and below this there was a round area of radio-opacity which on the lateral film appeared to be due to an exudate in the oblique fissure. Treatment with penicillin and sulfadiazine was started on July 20, and continued until August 3, 1945. The sputum was repeatedly negative for acid-fast bacilli. Repeated aspirations of the chest failed to reveal the fluid noted on the roentgenograms. Because the patient's temperature had become normal and he was asymptomatic by July 22, further aspirations were not attempted. On Sept. 6 the cavity could no longer be visualized but in the last chest roentgenogram of Sept. 6, 1945 the radio-opacity in the right lower lobe was still present. He was ambulatory, afebrile and asymptomatic when he was discharged on Oct. 14, 1945.

Comment. This patient was believed to have had a ruptured putrid lung abscess and an interlobar empyema. Under penicillin and sulfadiazine therapy he became asymptomatic and the cavity disappeared but the interlobar shadow persisted. We have had no opportunity to follow his progress, but believe that he will need surgical care for the residual lesion.

Case 5. W. B., a 28 year old negress, was admitted to Harlem Hospital on July 20, 1945. She had had no previous illness, operation or injury. She complained of cough and expectoration of four weeks' duration. She felt weak and had lost 15 lbs. On July 20 she noted pain in the right lower chest which was aggravated by respiration and cough.

On admission her temperature was 102° F., pulse 90 and respirations 20 per minute. She coughed and expectorated foul sputum. The chest film revealed consolidation at the right lower lobe just lateral to the heart. Her Wassermann reaction was positive. Her sputum contained no acid-fast bacilli, but aerobic and anaerobic gamma streptococcus, *Staphylococcus albus* and *B. subtilis*. The temperature continued until July 25 when treatment with penicillin and sulfadiazine was started and continued until August 7, 1945. Her temperature became normal on July 28 and all

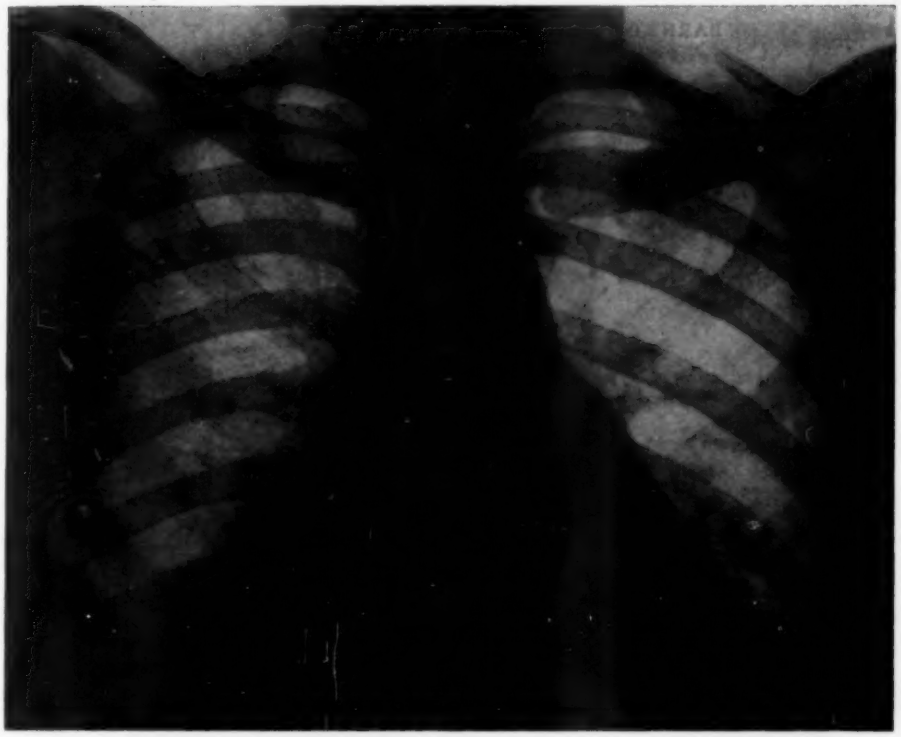


FIG. 4. *Case 5,* W. B. *A.* Film taken July 24, 1945 showing consolidation in the right lower lobe just lateral to heart.

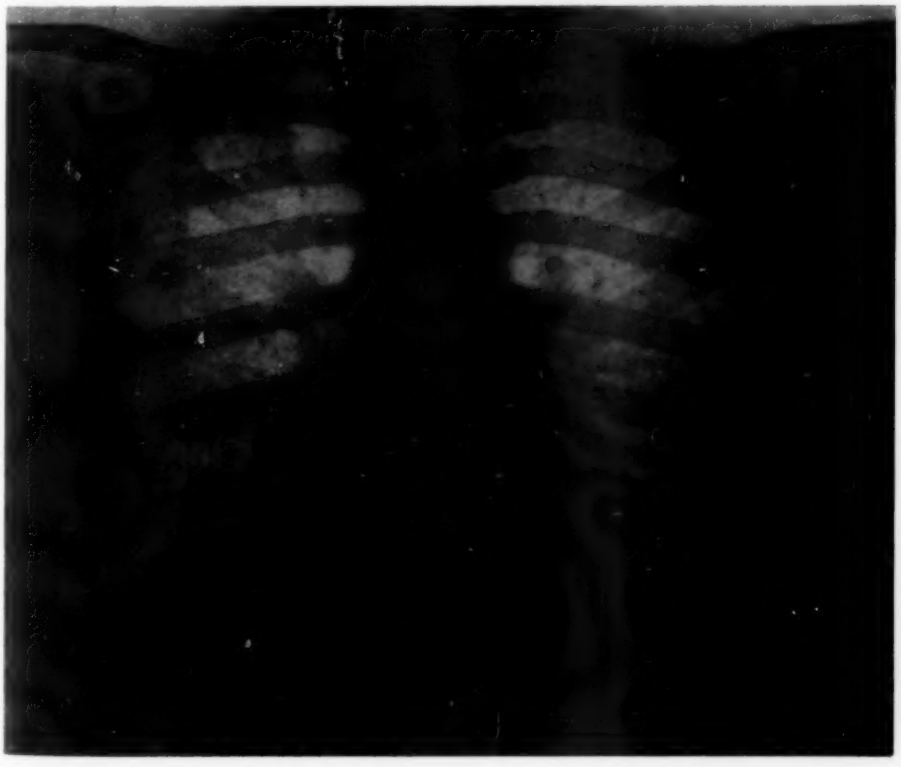
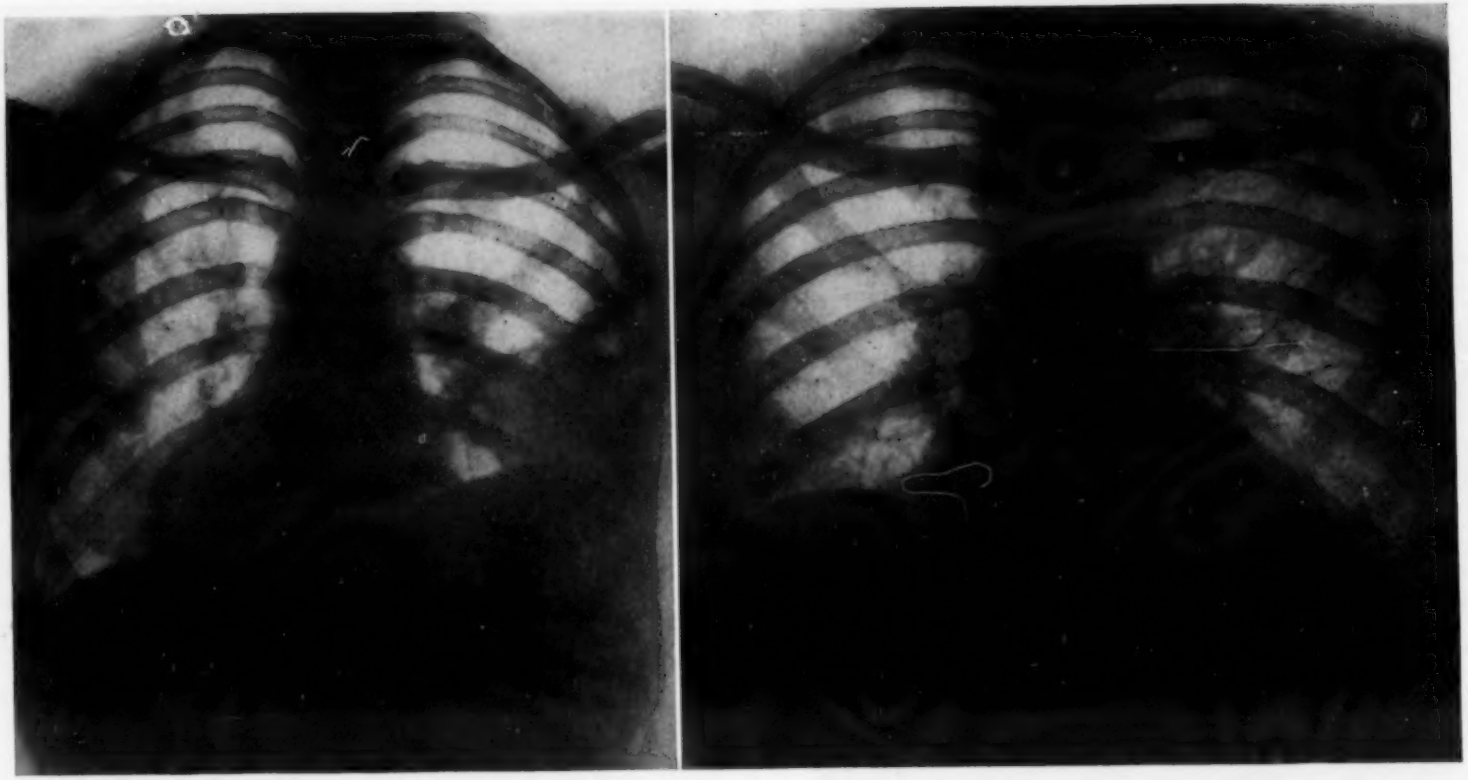


FIG. 4. *Case 5,* W. B. *B.* Film taken Aug. 20, 1945 showing clearing of lesion.



A B

FIG. 5. *Case 6*, H. H. *A*. Film taken Oct. 29, 1945 showing consolidation in the right lower lobe with cavity and fluid level. *B*. Film taken Dec. 5, 1945 showing clearing of cavity and consolidation. There is some pleural thickening at right base.

symptoms had disappeared by July 30. On August 20 her chest roentgenogram revealed no abnormality. A bronchogram on August 22 failed to reveal any abnormality. She was discharged on August 25, 1945.

Comment. This patient had an early acute putrid lung abscess which healed promptly with penicillin and sulfadiazine therapy.

Case 6. H. H., a negress 37 years of age, was admitted to Harlem Hospital on Oct. 7, 1945. On Sept. 30, 1945 she had had a peritonsillar abscess incised. Two days later her temperature began to rise and her physician suggested hospitalization.

On admission her temperature was 105° F., pulse 120, respirations 24. Her teeth were carious. She had marked trismus of the jaw. The anterior pillar of the left tonsil was edematous and intensely red. The right anterior pillar had been incised and both tonsils were markedly enlarged. Small moist râles were heard over the right anterior chest.

On Oct. 14 the temperature again rose and reached 104° F. on Oct. 17. At this time flatness, diminished breath sounds and tenderness were elicited over the right lower lobe and a large radio-opaque patch was noted in the region of the right lower lobe on the chest film.

On October 29 the chest film showed a large cavity with a fluid level in the region of the right lower lobe. The septic temperature continued until Nov. 10, 1945 and she was then transferred to the chest service where a diagnosis of putrid lung abscess was made. With penicillin and sulfadiazine her temperature became normal on Nov. 15, 1945. On Nov. 29 the chest film no longer showed a cavity, but slight pleural thickening was present in the region of the previous radio-opacity.

Comment. This patient made a remarkable recovery from her fetid abscess of the lung. Whether or not it could have been prevented by a longer period of treatment with penicillin when she was first admitted, it is difficult to say. In the light of our present experience we would administer penicillin in such cases not only until toxic symptoms disappear but until the roentgenograms show complete clearing of the pulmonary process.

Summary. Six consecutive patients with acute putrid lung abscess were treated with penicillin and sulfadiazine. All became free from local and constitutional symptoms. They improved remarkably in weight and strength. In five there was complete recovery from the disease as judged from the chest roentgenograms. In one a previously noted encapsulated exudate remained symptom-free and unchanged. The almost moribund patient with multiple bilateral abscesses and widespread bilateral pneumonia made a remarkable recovery and was left with only slight asymptomatic bronchiectasis. Four of the patients were admitted with large abscesses and grave toxic symptoms and formerly would have fallen into the group requiring immediate surgical care. One would probably not have survived operative interference. On penicillin and sulfadiazine they made a complete recovery without operation. There were no complicating local or metastatic infections.

Chronic Putrid Lung Abscess

During the past year, seven patients with chronic putrid lung abscess were admitted to Harlem Hospital. One of these had formerly been oper-

ated on in another hospital. He came in with convulsions and died the same day of a ruptured brain abscess.

One had a large putrid right upper lobe abscess with a spillover to the lower lobe of about six months' duration. Another had had a large putrid abscess in the left lower lobe for about three months. The fourth developed a putrid lung abscess in the right lower lobe following operation for ruptured gastric ulcer a year prior to admission to the Harlem Hospital where a spillover to the left side was noted.

The fifth patient was seen about six months after she developed an abscess in the right lung following a right "pneumonic" process. The sixth patient had several large foul lung abscesses in the right lower lobe for six months prior to admission to our wards, and the seventh had a large foul abscess in the right upper lobe for at least eight months before he came under our observation.

In five of these the treatment with penicillin and sulfadiazine was followed by unmistakable improvement. There was remarkable amelioration of the toxic manifestations. Serial chest roentgenograms disclosed definite regression of the lesion in two and marked improvement in one. There were no septic or metastatic complications while under treatment and in our opinion all of them became better surgical risks after penicillin-sulfadiazine treatment than they were before the treatment was instituted. One patient died of ruptured lung abscess one month after the treatment with penicillin was discontinued.

Discussion and Summary

In the past few decades there have been many reports of cures of isolated cases of acute putrid lung abscess with various chemicals, vaccines, and bacteriophage. Even artificial pneumothorax, obviously contraindicated in the treatment of such cases, came in for praise by some observers. The fact that the reported successes have not been reproduced with the respective methods of care in any fair group of patients with lung abscess suggests that the patients reported might have recovered spontaneously without treatment.

In the past year Roberts,[4] Dawson and Hobby,[5] Snook,[6] and Smyth and Billingslea[7] reported isolated cases of acute putrid abscess that recovered completely with penicillin therapy. The complete recovery of five of our six patients with such abscesses under the combined penicillin and sulfadiazine administration prompts the suggestion that this method of treatment deserves serious consideration and further extended trial, particularly since four of the patients belonged to a group heretofore considered in need of immediate surgical interference, one of whom would probably not have survived operation.

It is different in the case of chronic lung abscess. Here the lung and bronchi are converted by the reaction to the putrid infection into a maze of

fibrosis, multiple hard walled abscesses, bronchiectasis, atelectasis, and chronic pneumonitis, so that restitution to the normal can not be hoped for with medical care or even with extensive surgery. At times nothing short of lobectomy or pneumonectomy will save the life of the patient. Nevertheless the combined penicillin and sulfadiazine treatment in such cases is of inestimable value. It lessens toxicity, prevents further septic and metastatic foci, clears the surrounding pneumonitis, and improves the general condition of the patient so that he can better withstand the extensive operation indicated to bring relief.

It is important to bear in mind the need of continuing this combined penicillin sulfadiazine administration in acute putrid lung abscess not only until all toxic and local symptoms have disappeared but until the chest film shows no abnormal shadows in the segment of the lung involved. In chronic putrid abscess this method of care should be started preparatory to surgical intervention and continued after operation until all toxic symptoms disappear.

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TREATMENT OF VARIOUS INFECTIONS WITH PENICILLIN X, WITH A PRELIMINARY NOTE ON THE VALUE OF PENICILLIN X IN SCARLET FEVER*

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Penicillin X is a fraction obtained from cultures of the same mold (*Penicillium notatum*) which produces penicillin G (or "regular" penicillin). It differs from penicillin G in the method by which it is extracted, and is usually found more abundantly in flask-grown penicillin. We have tested the relative sensitivity of various bacteria to penicillin X and penicillin G and have found that two to 16 times as much penicillin G is required as penicillin X, unit for unit, to kill many bacteria in vitro. The details of this study will be published elsewhere. Other investigators [1-3] have reported similar results. In the present paper we are summarizing the results of the treatment with penicillin X of 104 patients suffering from various diseases.

Material and Methods

The penicillin X † used in this study was the calcium salt. The first batches of penicillin contained about 75 per cent of the X-fraction, and the later batches, 90 per cent. At first we used doses varying from 5,000 to 15,000 units every two or three hours. After studying the serum penicillin concentrations obtained with various doses, we decided upon 50,000 units every six hours as the dose which required a minimum of injections to obtain adequate serum concentrations. Figure 1 compares the mean penicillin concentrations obtained following the intramuscular injection of 50,000 units of penicillin X and crystalline penicillin G. Determinations were made on the sera of at least eight patients receiving each type of penicillin for each time interval. Although the serum concentrations are the same for both types of penicillin one hour after the injections, the serum concentrations are higher for penicillin X than for the penicillin G at similar intervals thereafter. Even more significant is the lack of detectable concentrations of penicillin three hours after an injection of crystalline penicillin G, whereas significant concentrations are obtained with penicillin X for at least six hours, and detectable levels for at least eight hours.

When continuous intramuscular administration was employed, the daily dose was 200,000 to 1,000,000 units, depending upon the severity of the

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disease. The drug was dissolved in 500 to 1,000 c.c. of isotonic sodium chloride solution using the method reported by two of us.[4] Penicillin X was also administered orally in tablets and capsules containing 25,000 units of the calcium salt and in aluminum hydroxide solution according to the method described by Welch.[5] The oral dose employed was 100,000 to 200,000 units every two hours.

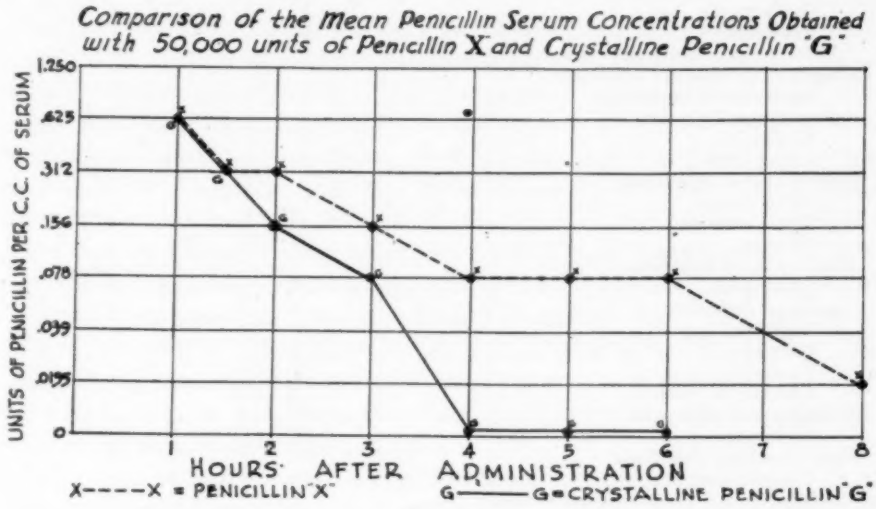

FIG. 1.

RESULTS

The diseases for which penicillin X was used may be divided into two groups (table 1). In Part 1 are tabulated the results observed in a variety of infections in which a specific organism was isolated from the patient. In Part 2 are listed the conditions in which the etiology was unknown.

A group of diseases caused by beta hemolytic streptococci was treated with penicillin X, including 34 patients with scarlet fever, seven with acute pharyngitis, two with suppurative otitis media, three with erysipelas, and two pharyngeal carriers. The use of penicillin in scarlet fever seemed indicated since hemolytic streptococci are highly susceptible to this drug. It was decided to treat with penicillin X all patients with scarlet fever whose temperature was 102° F. or above, who showed evidence of marked toxicity, or who presented complications. Several different dosage-schedules were employed, with 50,000 units every six hours established as the routine dose after trials of 5,000 to 15,000 units every three hours, and single daily injections of 100,000 to 300,000 units. Nearly all patients were treated for five days except a few in whom treatment was continued for several days longer because of persistence of complications present on admission.

The ages of the patients varied from one to 29 years. The number of cases occurring in both sexes and in the white and colored races was essen-

TABLE I

Results of the Treatment of Various Diseases with Penicillin X

	Recovered	Unimproved	Died
Part 1—Infections of Known Etiology			
Beta hemolytic streptococcic Infections			
Scarlet fever	33	1	
Streptococcus sore throat	7		
Otitis media	2		
Erysipelas	3		
Streptococcus carriers (pharyngeal)	2		
Streptococcus viridans Infections			
Bacterial endocarditis	1		2
Pneumococcic Infections			
Meningitis	2*		2*
Pneumonia	4		
Staphylococcic Infections			
Otitis media	2		
Otitis externa		1	
Abscess		1	
Meningococcic Infections			
Meningitis	2*	1†	
Gonococcic Infections			
Vaginitis	2		
Cervicitis	2‡		
Urethritis	2‡		
Chronic arthritis		1	
H. Influenzae Infections			
Meningitis	2§		
Fusospirochetal Infections			
Vincent's angina	1	1	
Part 2—Infections of Undetermined Etiology			
Infections of Ear, Nose, and Throat			
Acute pharyngitis	5		
Catarrhal otitis media	3		
Acute sinusitis	1	1	
Infections of the Skin			
Cellulitis	6		
Acne vulgaris		1	
Abscess	1		
Miscellaneous Diseases			
Bronchiectasis		1	
Bronchial asthma		1	
Infectious mononucleosis		1	
Typhoid		1	
Inclusion blennorrhea		1	
Mikulicz's syndrome		1	
Rheumatoid arthritis		1	
Epidural abscess and meningitis			1
Purulent meningitis	1		
Total	84	15	5

* Sulfonamides administered concomitantly.

† Sulfonamides administered concomitantly, meningococcic antiserum administered later in the course.

‡ Patients had acute arthritis also, which did not improve under treatment.

§ Sulfonamides and influenzal antiserum administered concomitantly.

tially the same. The admission temperature was 102° F. or over in all patients except five. Three of these five patients had complicating impetigo or abscesses on the hand or foot. The other two patients appeared markedly toxic although their temperatures were slightly below 102° F.

Of the 34 patients so treated, one failed to recover on this therapy alone. Although she received penicillin X for 72 hours, no improvement occurred until 18,000 units of antitoxin were administered. Two patients, who had received single injections of 200,000 and 300,000 units, respectively, improved initially but suffered a recurrence of pharyngitis on the third and eighth hospital days. One made an uneventful recovery after another dose of 200,000 units of penicillin and the other recovered on symptomatic measures. A third patient, who was treated with 50,000 units every six hours, developed a hemolytic streptococcic pharyngitis two weeks after penicillin was discontinued. This disappeared without treatment in two days. No other complications occurred in any of the patients who were given more than one injection. Two of the patients developed sequelae, namely, acute rheumatic fever and acute serous meningitis.[6] These patients ultimately recovered. In the remaining patients the temperature fell and symptoms disappeared soon after the penicillin X was begun. The time required for the temperature to fall and remain below 99° F. orally or 100° F. rectally, exclusive of fever due to complications, was calculated for each patient. It varied from four to 96 hours, averaging 54 hours. These figures do not include two patients, one of whom was given antitoxin, and the other had a normal temperature on admission and was treated because of the presence of an infected finger.

Nine patients entered with preëxisting complications, consisting in most cases of otitis media and cervical adenitis and infections of the hands or feet. All these complications cleared on penicillin therapy.

The response of patients with other hemolytic streptococcic infections treated with penicillin X was equally as good. The seven patients with acute sore throats exhibited rapid disappearance of fever and local symptoms. Hemolytic streptococci disappeared from their throats within 24 hours after treatment was begun and did not return while the patient was under observation. Similarly, two carriers of beta hemolytic streptococci showed prompt disappearance of the organisms within 24 hours after penicillin X therapy was started. In two patients with purulent otitis media all drainage ceased within 72 hours, and after treatment for seven and nine days, respectively, all evidences of infection had disappeared. There were three patients with erysipelas. They showed a prompt fall in temperature after penicillin was started. The lesions spread slightly during the first 24 hours, but then regressed and healed completely within five to nine days.

Three patients with *Streptococcus viridans* bacterial endocarditis were included in this series. One was treated for 19 days with 200,000 units of penicillin X daily by continuous intramuscular administration. He was admitted with a hemiplegia and known to have neurosyphilis. While under treatment, he developed evidence of a left lower lobe pneumonia and embolization of the right brachial artery. Serum penicillin concentrations were bactericidal for the organism and the blood stream was promptly sterilized, but the patient did not improve and died after 19 days of penicillin treatment.

At autopsy, large friable vegetations were found on a cusp of the aortic valve with perforation of the cusp. The aorta showed syphilitic changes and calcification. There was also evidence of a left lower lobe pneumonia and an embolus in the right brachial artery.

The second patient with endocarditis was a 31 year old colored male who had been ill for five weeks with constitutional symptoms and low grade fever. He had evidence of rheumatic disease with involvement of the mitral and aortic valves. After the organism was isolated, the patient was started on 200,000 units of penicillin X by continuous intramuscular administration. Although the blood stream was apparently sterilized, the patient continued to be febrile and the dose was increased to 500,000 units daily. Fourteen days after treatment was started, the patient had a sudden bout of respiratory distress and died. At autopsy there was evidence of an old active rheumatic myocarditis and endocarditis with rupture of several chordae tendineae of the mitral valve and evidences of congestive heart failure. A healing endocarditis was present on the mitral valve.

The third patient with bacterial endocarditis was treated for eight weeks with 50,000 units of penicillin intramuscularly every six to eight hours. He was admitted with a history of an influenza-like illness of two weeks' duration and a positive blood culture for *Streptococcus viridans* at another hospital. He had findings consistent with rheumatic heart disease with mitral valvulitis. Treatment has now been discontinued for one week and there is no evidence of infection.*

Pneumococci were the etiologic agents in 10 cases. Four patients had meningitis caused by pneumococci of Types 2, 3, 6, and 12 respectively. These patients received 200,000 to 500,000 units daily by continuous intramuscular administration with intrathecal injections of 20,000 units every 12 or 24 hours. In addition, the patients were given sulfonamides in large doses, orally or subcutaneously. In spite of these vigorous measures, two patients died. The spinal fluid was sterilized in both patients before death. Recovery was protracted in a third patient, a 52 year old male admitted in coma. He was found to have bilateral otitis media for which myringotomies were performed. The fourth patient was treated for one day with crystalline penicillin G. Therapy was changed to penicillin X when the infecting pneumococcus was found to be more sensitive to penicillin X than G in vitro. Although clinical improvement and clearing of the spinal fluid were gradual, she apparently recovered completely.

An infant with Type XIV pneumococcic pneumonia recovered coincidentally with penicillin X therapy after she had shown no response to sulfadiazine. Three adult patients with lobar pneumonia were treated with 15,000 units of penicillin X every three hours. Pneumococci, Types 1, 7, and 14, respectively, were isolated from the sputa of the three patients. Treatment was continued for at least 48 hours after the patients were afebrile.

* The follow-up period on this patient is now seven months during which time he has remained well.

The total duration of treatment was seven to 10 days. One patient had involvement of a whole lung plus delirium tremens. All patients made an uneventful recovery.

There were two patients with suppurative staphylococcic otitis media. The response was similar to that reported above in streptococcic otitis media. A patient with chronic bilateral otitis externa of *Staphylococcus albus* origin failed to improve after one week of penicillin X therapy. A hemolytic *Staphylococcus aureus* infection of the scalp with multiple abscesses was treated with penicillin X after sulfonamides and local measures had failed. There was no improvement after one week of treatment with penicillin X or after a longer course of penicillin G which followed.

Three patients with meningococcic meningitis were started on parenteral injections of penicillin X. Sulfadiazine was continued on two patients who had received the drug prior to admission. Both patients made an uneventful recovery, although this outcome might well have been expected from treatment with the sulfonamide alone. The third patient was given sulfonamides and penicillin X on admission because he had been in coma for several days and the prognosis seemed to be extremely grave. Meningococcic antiserum was administered several days later when no improvement was apparent. Death occurred one week after admission.

Gonococci were isolated from seven patients treated with penicillin X. Two children with vaginitis recovered on relatively small doses. Two patients had acute urethritis and complicating arthritis. The urethritis responded to therapy with penicillin X while the arthritis was not affected. On the other hand, in two patients with cervicitis and arthritis, both conditions disappeared after the same treatment. The drug was given to a patient receiving fever therapy for chronic gonococcic arthritis with no apparent effect.

Penicillin X was employed in two cases of *H. influenzae* meningitis in addition to sulfonamides and antiserum. No additional benefit appeared to result from the use of the penicillin X. The bacillus in each case was found to be highly resistant to penicillin in vitro.

Two cases of Vincent's angina were treated with intramuscular penicillin X. One patient showed no improvement with a persistence of the organisms after a single dose of 100,000 units. She recovered later on local measures. The second patient received two 24 hour courses of 300,000 and 100,000 units, respectively, with a one-day interval between courses. Local symptoms improved and the organisms disappeared from the lesions after the first course. However, local therapy was needed to clear the lesions completely.

A group of patients with acute pharyngitis, catarrhal otitis media and acute sinusitis, from whom a definite organism was not isolated, were among the patients treated. All patients recovered promptly except one with acute sinusitis who improved temporarily while on penicillin treatment, but had a return of symptoms the day following discontinuance of the drug.

Six patients with severe cellulitis of the face secondary to trauma, insect bites, infected teeth, impetigo, or infected herpes zoster also recovered.

Penicillin X was used in an attempt to abort an abscess which had developed at the site of an insulin injection. The penicillin X apparently caused an early localization, since on incision only a small amount of sterile seropurulent material drained. The wound healed rapidly after several days more of penicillin X therapy.

A group of patients with a variety of diseases such as acne vulgaris, bronchiectasis, bronchial asthma, infectious mononucleosis, typhoid fever, inclusion blennorrhea, Mikulicz's syndrome, and rheumatoid arthritis did not improve on doses of penicillin X which were adequate for other infections.

Penicillin X was used in two patients with meningitis secondary to bilateral otitis media from whom no causative organism was isolated from the ears or spinal fluid. One patient recovered on the regime previously described for pneumococcic meningitis plus bilateral myringotomy. In the second patient the otitis media and meningitis cleared after treatment with massive doses of sulfonamides plus parenteral penicillin, but he died following an operation, which included bilateral mastoidectomy and evacuation of an epidural abscess. Autopsy revealed a large cerebellar abscess in addition.

No toxic effects were observed in any patient receiving penicillin X. There were no untoward effects noted after the injection of penicillin X into joint and pleural spaces or the intrathecal space. An occasional patient complained of slight pain at the site of an intramuscular injection.

TABLE II

Comparison of the Course of Scarlet Fever in Patients Receiving Penicillin Compared with Those Treated by Other Measures

Group	Number of patients	Total duration of fever (hours)	Number of patients developing complications after admission
A	34	54	3
B	35	71	11
C	13	74	2
D	34	99	7

Group A—Patients received penicillin X.
Group B—Patients admitted during the same period and treated symptomatically because of the mildness of the disease.
Group C—Patients admitted during the same period and treated symptomatically, or with sulfadiazine for complications, because of delay in diagnosis.
Group D—Patients treated with sulfadiazine and/or antitoxin prior to the period of the use of penicillin.

COMPARISON OF DIFFERENT METHODS OF TREATING SCARLET FEVER

In table 2 we have compared the results of the treatment of our patients with scarlet fever, who received penicillin X (Group A), with the results in three other groups of patients. Included in Group B are all the patients

admitted during the same period as Group A, but who did not receive penicillin X because they did not fulfill the criteria established for penicillin therapy. Group C consists of 13 patients eligible for penicillin, but in whom the diagnosis was not established until the day after admission, and who were, therefore, treated only symptomatically or given sulfonamides if a complication developed. A group of 34 patients with findings similar to those in Group A, who were admitted in the months preceding the use of penicillin, was selected for comparison and is included in Group D. We have compared the total duration of fever in all four groups and found it to be the shortest in the penicillin-treated group, averaging 54 hours. These results compared favorably with 71 hours for the milder untreated patients in Group B and 74 and 99 hours for patients in Groups C and D in whom the illness was as severe as in the patients in Group A. The patients in Group D were admitted during the winter months when the disease is ordinarily more severe, which may account for some of the difference. However, patients in Group C were admitted during the same season as Group D patients and the results are significantly different from the results in Group A, even though the number of patients is admittedly small.

A significant evaluation of the therapy is a comparison of the number of patients developing complications in each group. In the penicillin-treated group only three patients developed complications, two of whom had been inadequately treated. The third patient had a pharyngitis which appeared two weeks after the completion of penicillin therapy and subsided spontaneously in two days. In the 31 remaining patients, who were adequately treated, no complications occurred. In Group B there were 11 patients with complications. Four patients in the group had received antitoxin and one sulfadiazine, and two of the antitoxin-treated patients were among the 11 with complications. Two patients in Group C developed complications and seven in Group D. Among the patients with complications in the last group, four had received either sulfonamides, antitoxin, or both, as did 14 other patients in this group.

Recently Meads and his co-workers [7] reported their results in a small series of cases of scarlet fever treated with penicillin and other forms of therapy. Their impression was that a more complete and rapid clinical cure occurred in the patients treated with intramuscular penicillin. They noted that although the course of the disease was not significantly altered, no septic complications occurred in this group.

Nine patients in the penicillin-treated group entered the hospital with complications already present. Similarly, five, two, and 12 patients had complications present on admission in Groups B, C, and D. Comparison of the time necessary for these complications to improve showed that the penicillin X-treated patients recovered more rapidly than the others, although the patients in the other groups were treated with sulfadiazine.

There was only one failure in the penicillin-treated group. When the patient showed no improvement after 72 hours of this treatment, she was

given 18,000 units of antitoxin and responded with a prompt drop in temperature and diminution in toxicity. It was later determined that the hemolytic streptococcus isolated from her throat was resistant to the serum penicillin concentrations obtained in the patient.

The ability of penicillin to inactivate the hemolytic streptococcus erythrogenic toxin was studied.[8] The results showed that the effectiveness of penicillin was not due to neutralization of the toxin.

Discussion

Penicillin X has been used successfully in the treatment of gonococcic [1] and other infections [2] which are susceptible to penicillin G. We have obtained good results in infections caused by the hemolytic streptococcus, pneumococcus, staphylococcus, and the gonococcus. There was no evidence that it produced any better results than penicillin G in these infections nor that it was effective in other diseases which do not respond to penicillin G. The only clinical evidence of the superiority of penicillin X over penicillin G was that a satisfactory therapeutic response could be obtained when injections of 50,000 units were given as infrequently as every six hours. These results may be explained by the fact that higher and more prolonged serum concentrations of penicillin were usually obtained from the administration of penicillin X than from equivalent doses of penicillin G.

The chief value of penicillin X may be found in infections in which the causative organism is relatively resistant to penicillin G and yet sensitive to penicillin X.

Further clinical observation on a larger number of patients is necessary before the relative value of the two types of penicillin can be completely evaluated.

Summary and Conclusions

1. One hundred and four patients with various infections were treated with penicillin X. The results were at least as good as when penicillin G was used.

2. Penicillin apparently decreases the length of the febrile period and reduces the number of complications in patients having scarlet fever.

3. A further trial of penicillin in scarlet fever seems warranted. Further observations are needed to evaluate the relative merits of penicillin X and G in this and in other infections.

We wish to thank the staff of the Georgetown Medical Division for the privilege of reporting several of these cases, and Miss C. Barbara O'Neil and Mrs. Rose Breen for technical assistance.

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SERUM AMYLASE AND SERUM LIPASE IN MUMPS *

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THE recent valuable reports of Zelman [1] and Applebaum [2] on serum amylase in mumps appeared while we were engaged in carrying on studies of mumps and its complications. We became particularly interested in testing whether or not elevated serum amylase values could be of assistance in aiding in the diagnosis of orchitis and epididymitis due to mumps when there was no history or evidence of parotitis.[3] The additional problem of attempting to determine the effect of mumps on serum lipase presented itself, for if, as some investigators [4, 5] believe, the serum amylase elevation noted in mumps is due to a silent pancreatitis, it would be reasonable to expect concomitant elevation in serum lipase. Zelman considered this last problem but abandoned it because of objections to the method ordinarily used to determine serum lipase.

The origin and function of serum amylase is not known.[6, 7] Ligation of the parotid duct results in an increase of serum amylase.[7] Pancreatic duct ligation is likewise followed by an elevation of serum amylase in the blood, but it returns to normal in 8 to 15 days.[6] The following conditions have also been found to cause elevated values of serum amylase: acute pancreatitis [2, 6, 7, 8, 9, 10]; perforation of peptic ulcer into or near the pancreas [6, 10]; trauma to pancreas (experimental) [7]; parotitis [1, 2, 10]; increased thyroid activity.[11] Age, sex, diet, vitamin deficiency and starvation do not affect serum amylase.[7, 10] Patients with liver disease have amylase values which tend to be low.[9, 10] Nevertheless, the liver contains no amylase.[7] Diseases of carbohydrate metabolism have normal values.[8] However, Heifetz et al. have reported that the diastatic activity of the blood is lower in the more severe diabetics.[10] Amylase (diastase) is excreted by the kidneys.[12] Thus impaired amylase excretion has been noted in renal insufficiency.[10, 13]

The method of Somogyi [14] was employed since it is the one standard at the Naval Medical School.[15] The principle involved is the hydrolysis of a starch solution by amylase in the serum. After incubation of the starch mixture for a definite period, it is added to an iodine solution to determine the end point for complete hydrolysis. The end point is an absence of blue. The results are reported in terms of units. The normal range is up to 320 units.

Fifty-four normal adults were used to determine the normal range. Table 1 shows the results obtained.

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TABLE I

Units	Frequency
228	24
266	10
320	18
400	2

Our results corresponded well with the usual values found.

The experimental group consisted of 224 mumps patients of whom 68 developed orchitis and epididymitis. Six hundred eighty-four serum amylase determinations were done. An initial determination was done usually on the day of, or on the day after admission. Each patient had a serum amylase test repeated approximately one week after admission and often an additional one on the day of discharge. The average period of hospitalization was terminated on the fourteenth day of disease. Some patients were carried over to the fifteenth to seventeenth day of disease owing to local regulations for days of discharge. Table 2, which follows, contains in the

TABLE II

Serum Amylase Determinations in Mumps

Serum Amylase in Units	Day of Disease*				
	1–7	8–14	15–21	22–32	Total
228	11	21	25	11	68
266	5	7	14	3	29
320	27	44	35	15	121
400	35	46	16	4	101
533	47	60	9	6	122
800	61	39	7	1	108
1600	79	51	5	0	135
Total	265	268	111	40	684
% Serum Amylase values above normal, i.e. 400 and over	83.77%	73.13%	33.33%	27.50%	

* Calculated from day of onset of symptoms, not necessarily from day of admission to the sick list.

column headed 15 to 21 day determinations obtained from some of these carry-overs, but the greater number is made up of determinations done on patients who had developed orchitis as a complication. In the fourth week and over group (22 to 32 day) appear values obtained almost entirely from patients who had orchitis as a complication. Of the 224 patients, 86½ per cent were 17 through 23 years of age; 13½ per cent were 24 through 39 years of age.

It may be seen from table 2 that during the first week of the disease, 83.77 per cent of all determinations show an elevated serum amylase. There were 43 patients who had normal serum amylase values in the first week of

the disease. Thirty-three of these 43 showed elevated values in the second week of the disease. Thus 96.22 per cent of all mumps patients had elevations of serum amylase some time during the first two weeks of the disease. This confirms the observations of Applebaum.[2] It may be that if more observations on serum amylase had been done on the 10 patients whose amylase values remained normal, elevations would have been recorded. Further study of table 2 shows that there is a progressive decrease in the levels of serum amylase from week to week.

Lewison [9] observed 13 cases of mumps. No effect was noted on the serum amylase of the patients who developed orchitis. Murphy et al.[16] reported a similar experience in their observations on seven cases of orchitis complicating 35 cases of mumps.

Table 3 analyzes the values of serum amylase in 68 cases of orchitis and epididymitis which complicated the 224 patients with mumps, under study. All the values noted are determinations made after the appearance of orchitis and epididymitis. The 68 cases are arranged in groups according to the day of onset of the complication, i.e. 1–7 day; 8–14 day; 15–17 day. No case of orchitis occurred later than 17 days after the onset of mumps. The serum amylase determinations are arranged in groups of weeks (1–7 day; etc.) according to the time the determinations were done in relation to the day of disease. These values are compared with determinations done on the cases of uncomplicated mumps plus the cases of mumps before the appearance of the complication.

It may be seen from table 3 that serum amylase values in mumps orchitis parallel the values in uncomplicated mumps. It is evident that the elevations of serum amylase are a function of the disease associated with the parotitis alone. Hence, as a clinical aid in establishing the etiology of an orchitis and epididymitis, the serum amylase would be of limited value. There are two types of orchitis and epididymitis due to mumps which may present themselves for diagnosis. The first type follows a parotitis which goes unnoticed by the patient and perhaps not observed by the examiner. Here, the value of serum amylase would depend upon how soon the orchitis developed after the onset of the missed parotitis. The later the orchitis developed, the less the chance that the serum amylase would be elevated. The second type is the true primary orchitis which occurs without the appearance of parotitis. In this second type serum amylase determination would probably be entirely normal. This will be discussed later after consideration of serum lipase in mumps. The finding of an increased serum amylase in a case of orchitis would point towards mumps as the etiologic factor. A normal serum amylase would not necessarily rule out mumps. One would then have recourse to other aids such as (a) careful history of contact, (b) lumbar puncture and cell count on spinal fluid.[3, 17]

A number of patients were encountered with conditions other than mumps in which there was swelling of the face or neck, often in the parotid region. Serum amylase determinations were made on them to determine

TABLE III

Analysis of Serum Amylase after the Appearance of Orchitis and Epididymitis

Day of Occurrence of Orchitis and Epididymitis	Number of Cases		Day of Disease			
			1–7	8–14	15–21	22–32
1–7	43	No. Amylase Determinations	38	50	38	22
		% of Values 400 Units or More	81.57%	58.0%	34.42%	28.51%
8–14	19	No. Amylase Determinations		16	16	(b) 8
		% of Values 400 Units or More		62.5%	31.25%	(50%)
15–17	6	No. Amylase Determinations			6	(b) 7
		% of Values 400 Units or More			0.0%	0.0%
Uncomplicated Mumps Including Mumps Cases before Development of Orchitis and Epididymitis	(a) 211	No. Amylase Determinations	227	202	51	(b) (c) 3
		% of Values 400 Units or More	84.14%	77.22%	37.05%	33.33%

(a) 13 cases were admitted with orchitis and parotitis.
(b) % calculated from these small groups are of very limited value.
(c) 3 patients with uncomplicated mumps, who developed mumps while convalescing from another condition.

the specificity of the elevations noted in mumps. Table 4 lists the conditions and the amylase findings.

TABLE IV

Other Conditions with Swelling of Face or Neck

Diagnosis	Number of Cases	Units Serum Amylase
1. Lymphadenitis secondary to scarlet fever	12	320 or less
2. Lymphadenitis secondary to tonsillitis	3	320 or less
3. Cellulitis of face following antrum perforation during treatment for sinusitis	1	266
4. Impacted molar tooth with swelling over mandible	1	200
5. Fracture of mandible with swelling in parotid region	1	320
6. Recurrent submaxillary gland swelling secondary to duct stenosis	1	320
7. Lymphadenitis secondary to scarlet fever	1	400
8. Lymphadenitis secondary to peritonsillar abscess	1	533
9. Faucial diphtheria with *"bull neck"*	1	400 400
10. Diphtheria with cervical lymphadenitis and slight soft tissue edema	1	533

Of 17 cases of lymphadenitis listed in table 4 only two cases showed elevated values. These determinations were not repeated, hence the two

elevations are open to some question. Of interest is the case of recurrent submaxillary gland swelling with normal serum amylase. The findings in our two cases of diphtheria were striking. More determinations are required of the conditions listed above to determine the specificity of serum amylase determinations in swelling of the face and neck. This is particularly true of diphtheria with edema of the face and neck ("bull neck"). Applebaum [2] noted three cases of extra-parotid mumps with normal amylase values. Two involved the submaxillary glands and one involved the sublingual glands. He explained these findings by pointing out that parotid saliva is particularly rich in salivary amylase as compared to the other salivary glands. Since the damming-back of amylase into the blood stream in cases of parotid inflammation is thought to be a mechanism by which the blood serum amylase is raised, high values would be expected in parotid mumps and low ones in submaxillary or sublingual mumps. Lewison [9] found normal serum amylase values in 94 per cent of 720 patients having clinical conditions other than mumps and diseases of the biliary system.

Serum lipase is not affected by food or starvation.[6] It is increased in pancreatitis,[6] duodenal ulcer perforating into the pancreas,[6] pressure of tumor arising in or near the pancreas such as enlargement of lymph nodes near the head of the pancreas,[18] pancreatic lithiasis in which a stone blocks one of the pancreatic ducts.[18] Lewison states that in his limited experience with the serum lipase test, its activity parallels that of serum amylase in pancreatic disease. But he states it takes 24 hours for the elevated lipase to appear in acute pancreatitis. This fact diminishes its usefulness in clinical emergencies.[9] Johnson and Bockus [18] emphasize the specificity of hyperlipasemia in pancreatic disease.

The serum lipase method employed in this study, recommended by the Naval Medical School,[19] is a modification of the method of Lovenhart by Cherry and Crandall as used by Comfort and Osterberg.[20] It depends on the estimation of fatty acid which results from the hydrolysis of an olive oil emulsion by the blood serum lipase which acts on the oil over a 24 hour period under fixed conditions of hydrogen-ion concentration and temperature. The fatty acid is titrated against N/20 sodium hydroxide. The normal range is given as 0.0 c.c. to 1.5 c.c. of N/20 NaOH.[6, 19] However, Johnson and Bockus using this method have found that the normal values are less than 1.00 c.c. N/20 NaOH.[21] In our study, normal men showed serum lipase values less than 1.00 c.c. of N/20 NaOH.

Serum lipase determinations were done on 54 normal men in the same age group as those patients who were treated for mumps. Table 5 records our findings.

The normal mean was found to be 0.309 ± 0.016 c.c. of N/20 NaOH with a standard deviation 0.184 ± 0.012 c.c. N/20 NaOH. The range is calculated to be 0.00 to 0.86 c.c. This upper limit of normal is closer to the value noted by Johnson and Bockus (i.e. value less than 1.00 c.c. for upper normal.[18, 21]

TABLE V

Serum Lipase in Normal Men

Serum Lipase in c.c. N/20 NaOH	Frequency
0.00	0
0.05	4
0.10	5
0.15	8
0.20	10
0.25	0
0.30	3
0.35	2
0.40	6
0.45	2
0.50	2
0.55	2
0.60	4
0.65	1
0.70	1

Table 6 was compiled from 671 determinations of serum lipase done on 224 cases of mumps.

TABLE VI

Serum Lipase in 224 Cases Mumps

Serum Lipase in c.c. N/20 NaOH	Frequency				Total
	Day of Disease				
	1–7	8–14	15–21	22–32	
0.00	3	12	2	2	19
0.05	6	3	2	0	11
0.10	46	24	26	7	103
0.15	23	16	6	3	48
0.20	45	42	13	6	106
0.25	14	24	4	1	43
0.30	27	30	12	3	72
0.35	7	9	5	5	26
0.40	21	25	6	3	55
0.45	10	7	3	3	23
0.50	14	12	6	3	35
0.55	7	9	4	2	22
0.60	18	12	6	0	36
0.65	2	5	6	0	13
0.70	3	7	5	1	16
0.75	0	1	1	0	2
0.80	2	6	0	0	8
0.85	0	1	0	0	1
0.90	4	1	2	0	7
0.95	0	1	0	0	1
1.00	5	2	2	1	10
1.05	0	0	0	0	0
1.10	5	4	0	0	9
1.15	0	0	0	1	1
1.20	1	2	0	0	3
1.25	1	0	0	0	1
Total	264	255	111	41	671

The average serum lipase with its standard deviation was calculated for each time unit noted in table 6. Table 7 contains a summary of these calculations as well as the average calculated from serum lipase determinations done on 54 normal men.

TABLE VII

Summary of Serum Lipase Determinations in Terms of c.c. N/20 NaOH Done on 54 Normal Men and 224 Patients with Mumps

		Number of Determinations	Mean	Standard Deviation
	Normal Group	54	0.309 ± 0.016 c.c.	0.184 ± 0.012 c.c.
Mumps (224 patients)	1–7 day	264	0.334 ± 0.010 c.c.	0.2439* ± .0068 c.c.*
	8–14 day	255	0.349 ± 0.010 c.c.	0.2438 ± .0072 c.c.
	15–21 day	111	0.333 ± 0.015 c.c.	0.234 ± 0.010 c.c.
	22–32 day	41	0.323 ± 0.024 c.c.	0.236 ± 0.017 c.c.
	Total Mumps	671	0.3365 ± 0.0062 c.c.	0.2412 ± 0.0044 c.c.

* Reasons for retention of decimals in published biometric-constants are set down by Raymond Pearl.[22]

The means noted in table 7 show relatively slight differences. The greatest difference lies between the normal mean 0.309 ± 0.016 c.c. and that noted in the group 8–14 day, 0.349 ± 0.010 c.c. x/d calculated for the difference between these two means was approximately 0.66. This denotes that there is no significant difference between them. The means of the remainder of the groups likewise showed no significant difference from the mean of the normal group.

The upper limit of serum lipase in our control normal group was 0.86 c.c. of N/20 NaOH. In the 671 determinations done on our mumps patients, there were 27 patients who showed serum lipase values of 0.90 to 1.25 c.c. of N/20 NaOH somewhere in the course of their illness. A careful review of the charts of these patients showed that not a single one had any evidence of acute pancreatitis. One patient had an episode of vomiting associated with fever at the onset of orchitis. The nurses' notes on another patient showed that he had an "upset stomach" on admission. Both of these patients were seen at least twice daily from the day of admission to the day of discharge and in neither was there any complaint of abdominal pain or evidence clinically of pancreatitis. Thus in the 27 patients (32 lipase determinations above 0.85 c.c.) with slightly elevated serum lipase values, there was no clinical pancreatitis present. In the remainder of the mumps patients with normal serum lipase there was likewise no case of clinical pancreatitis.

Hyperlipasemia is said to be specific for acute pancreatitis.[19] Values as high as 12.0 c.c. of N/20 NaOH have been reported in acute pancreatitis.[6]

Johnson and Bockus have found abnormal values ranging from 1.0 to 10.0 c.c.[18] Hence, since our upper normal was 0.86 c.c. of N/20 NaOH, the maximum number of patients who could have had a silent pancreatitis was 27. We must conclude, therefore, that "silent pancreatitis" if it exists at all, is the exception and not the rule.

The great majority of the 224 patients under study showed an elevation of serum amylase with normal serum lipase. It must hold then that these elevations of serum amylase are extra-pancreatic in origin. This is likewise borne out by other facts such as (a) the finding of an elevated serum amylase in

(1) Ligation of the parotid duct.[7]
(2) Calculous obstruction of the salivary duct.[10]
(3) Suppuration of the salivary gland.[10]

(b) The finding of normal amylase values in extra-parotid mumps involving the submaxillary or submental glands.[2]

To return to a problem mentioned above, it is evident that, in primary orchitis and epididymitis unassociated with involvement of the parotid gland but secondary to systemic mumps, the serum amylase determination would be expected to fall in the normal range. An elevated serum amylase would point to a missed parotitis with systemic mumps.

Conclusions

1. Serum amylase determinations are of value in the differential diagnosis of parotitis.

2. 96.22 per cent of 224 patients with mumps showed elevations of serum amylase in the course of the disease.

3. In the first week of disease 83.77 per cent of the mumps patients showed elevations of serum amylase. This percentage fell progressively week by week so that in the fourth week (actually 22–32 day) 27.5 per cent of patients still showed elevated values of serum amylase.

4. Elevations of serum amylase apparently follow the evolution of the parotitis.

5. The appearance of orchitis and epididymitis as complications of mumps does not affect the amylase of the serum. Hence in apparently primary acute orchitis or epididymitis of mumps origin, the elevation of serum amylase will depend upon how soon the orchitis and epididymitis follow the original parotitis (which may have been overlooked by the patient or missed by the examiner). In true primary orchitis and epididymitis of systemic mumps origin unassociated with parotitis, normal values of serum amylase may be anticipated.

6. Elevated serum amylase found in uncomplicated mumps is extra-pancreatic in origin.

7. 89.5 per cent of 224 mumps patients had normal serum lipase values. 11.5 per cent of patients had slightly elevated values. None was greater

than 1.25 c.c. of N/20 NaOH and none showed any clinical evidence of pancreatitis.

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THE PROTHROMBINOPENIC EFFECT OF MASSIVE SALICYLATE THERAPY IN ACUTE RHEUMATIC FEVER *

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FOLLOWING the appearance of Coburn's report [1] advocating large doses of salicylates in rheumatic fever, interest in the problem of salicylate intoxication has increased and warnings have been sounded regarding the dangers of salicylate therapy.[2, 3] Stress has been laid upon hemorrhage as one of the most striking of these complications, and case reports are cited of deaths from hemorrhage, but prothrombin levels were not determined in any of the fatal cases reported.[4, 5]

Quick questioned the rôle of salicylate hypoprothrombinemia in the production of fatal hemorrhage, on the grounds that no evidence had been offered that prothrombin levels could be sufficiently reduced by salicylates to cause hemorrhage.[6] Recently, Fashena and Walker [7] reported prothrombin times of 60 to 90 seconds in six children treated with large doses of salicylates, but made no mention of hemorrhage occurring in these patients.

It is considered of interest, therefore, to report the following study, which was undertaken to determine the behavior of prothrombin in adults treated with large doses of salicylates.

The subjects were 25 cases of acute rheumatic fever, treated by Coburn's method. Their ages ranged from 18 to 40. All had been hospitalized for rheumatic fever in an acute phase.

METHODS

The patients were given 10 grams of sodium salicylate in 1,000 c.c. of normal saline intravenously over a four hour period daily for six days or longer, depending on the patient's clinical course. Those patients who failed to show a prompt response in symptoms, fever, and sedimentation rate were given an additional 10 grams of sodium salicylate daily. Thereafter, 10 grams of the drug were given orally each day in divided doses at four hour intervals. Duration of treatment varied from 21 to 60 days depending on the patient's response to therapy.[1] Daily physical examinations were made, a four hour temperature chart was kept, erythrocyte sedimentation rate (Cutler), complete blood count and urinalyses were determined at least every third day.

The Magath modification of the Quick method of determining the prothrombin time was used.[8] Thromboplastin was obtained from fresh rabbit brain; each prothrombin time determination was checked by a normal con-

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trol. By this technic, normal plasma clots in 18 to 22 seconds. In each instance, the prothrombin time was obtained before salicylates were administered, and was repeated at least every third day while the patient was undergoing treatment.

Quantitative blood salicylate analyses were made at three day intervals throughout the period of therapy.[1] Determinations were made in the mornings prior to any intravenous salicylate administration and so during the period of intravenous therapy represented the drug concentration residual after the previous day's administration. The levels obtained during oral therapy were uniformly higher than the residual levels during parenteral administration. Concentrations of drug were maintained at about 35 mg. in almost all cases. In only three cases was the maximum salicylate concentration under 40 mg. per cent; in 18 the levels ranged from 40 to 49, and in six the levels exceeded 50.

Because of the known effect of liver disease on the prothrombin level of the blood,[9] efforts were made to eliminate this factor. No history suggestive of hepatic disease was obtained from any of the patients studied. Cephalin cholesterol and intravenous hippuric acid tests were performed on each patient before and after salicylic acid therapy with entirely normal results.

Results

Two effects of massive salicylate dosage on the blood prothrombin were noted. A moderate reduction of prothrombin to 55 to 75 per cent of normal occurred in all cases after the third or fourth day of treatment. In addition, a maximum effect, of short duration, was observed to occur in a number of cases. The range of this peak effect is indicated in table 1.

Table I

Maximum Prothrombin Depression (Percentage of Normal)

Prothrombin	10–19%	20–29%	30–39%	40–49%	50–59%	60–100%
No. of Cases	2	10	4	5	6	0

It will be noted that two cases fell below the critical level of 20 per cent prothrombin, and 12 cases, or almost half the total, reached levels below 30 per cent of normal.

In two cases a second course of treatment was required. In both instances the hypoprothrombinemia was less than that observed during the original course of massive therapy.

Of interest was the variation in the time of appearance of the maximum hypoprothrombinemic effect in the series (table 2).

Table II

Time at Which Maximum Reduction of Prothrombin Occurred

Max. Hypoprothrombinemia	1st Wk.	2nd	3d	4th	5th	6th Wk.
No. of Cases	1	9	9	1	3	4

The duration of pronounced prothrombin depression was observed to be from one to three days in all cases except two in which the effect was greatest. In these two cases, the marked prothrombin depression lasted from three to six days. Despite continuation of salicylates, there was a spontaneous and rather rapid return of the prothrombin time toward normal levels. This tendency, so marked following the maximum depression of prothrombin, was noted as a general trend following the third week of therapy, when the prothrombin time approached normal values despite the maintenance of high levels of salicylate concentration in the blood (chart 1).

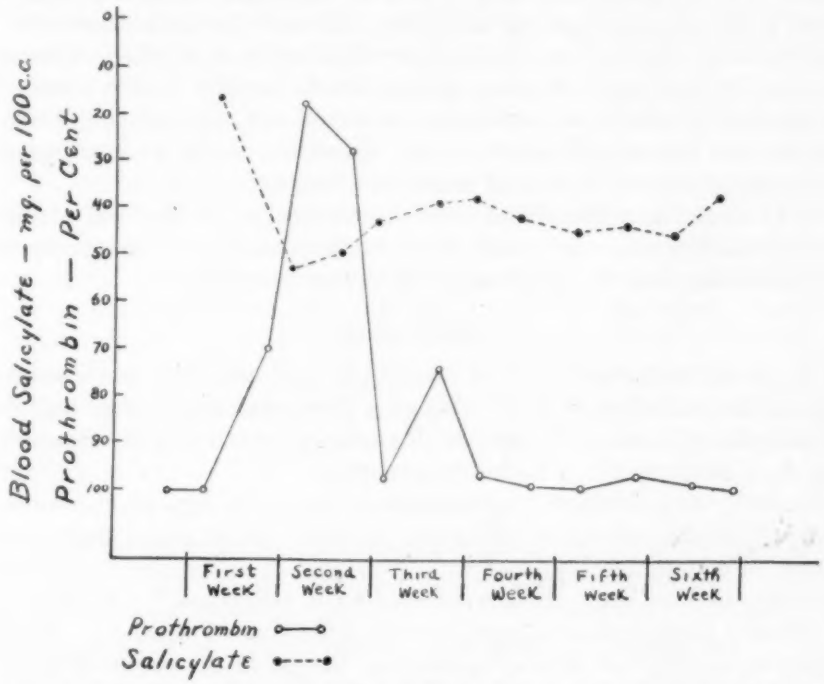

CHART 1. Relation of blood salicylate level to the degree of prothrombin deficiency. (Illustrative case)

The exception to this trend was in those cases in which the maximum hypoprothrombinemia was late in appearing (seven cases). These, however, showed the same trend toward normal levels of prothrombin concentration at a similar interval following the peak effect.

Evidence of bleeding was sought carefully in each case. Five patients developed epistaxis, which was severe in only one case. Two of the five also showed small splinter hemorrhages under the finger nails. These hemorrhages have been observed by us and others in patients undergoing dicoumarol therapy. The patient with severe epistaxis had a prothrombin percentage of 24 per cent of normal. Those with both epistaxis and splinter hemorrhages had prothrombin percentages of 16 and 22 per cent at the time

the bleeding occurred. In the remaining cases of epistaxis, prothrombin percentages ranged from 21 to 28 per cent of normal. In all instances, salicylate therapy was continued, and the bleeding was controlled by ordinary measures. Spontaneous return of prothrombin time to normal was observed in these patients as well as in all others in the group. No instance of bleeding was observed after the third week of treatment.

In a series of rheumatic fever patients treated by penicillin, without salicylates, and under rigid controls, no hypoprothrombinemia nor bleeding was observed.

In brief, hypoprothrombinemia of some degree occurred early in all cases treated with massive doses of salicylate. A more marked depression was noted in some cases. This effect occurred abruptly, was of short duration, returning spontaneously toward normal levels usually within three days. The marked prothrombin depression occurred any time during treatment from the first through the sixth week. However, in the majority of cases, it occurred during the first three weeks of treatment.

In 12 cases the prothrombin level dropped below 30 per cent of normal. In five cases bleeding, consisting of epistaxis or small nail bed hemorrhage, occurred at the time of maximum prothrombin depression.

Discussion

The prothrombinopenic effect of salicylic acid was first demonstrated by Link and his associates [10] in the rat, after their chemical studies had shown that salicylic acid was an important degradation product of the hemorrhagic agent 3, 3′ methene bis(4 hydroxycoumarin).[11, 12]

Meyer [13] and Shapiro [9] independently reported hypoprothrombinemia following administration of salicylates in man. Both found that synthetic vitamin K counteracted this effect. Rapoport et al. reported prothrombin depression in rheumatic children treated with salicylates.[14] In their series the greatest prolongation of prothrombin time was 35 seconds. However, Coburn in his original series, reported no change in the prothrombin time.[1] More recently others have reported complete absence of prothrombin effect in salicylate treated patients.[15] Possible explanation for these divergent results is offered by the present series of cases in which the marked hypoprothrombinemia was found to be of short duration, and occurred at almost any time during the course of treatment.

Butt's study [16] in which he reported uniform but only moderate prothrombin depression by salicylates is difficult to compare with the present study. His patients were given gradually increasing doses of salicylates, rather than massive doses at the outset, as in the present series. Moreover, the majority of Butt's patients were convalescent, whereas all our patients were in an acute phase of rheumatic fever with possible depletion of their vitamin K stores as a consequence.

This latter factor may account for the more profound prothrombin depression noted in some of our patients, as well as for the individual variation

in time of occurrence and degree of severity of the marked prothrombin depression.[17]

The five cases reported here of bleeding occurring at the time of maximum hypoprothrombinemia induced by salicylates, are the only ones thus far reported to our knowledge. Moreover, it must be stressed that in these cases the prothrombin returned spontaneously to normal levels during continued administration of salicylates, and with its return, the bleeding tendency stopped. The uniformity with which this occurred, together with the general tendency, noted above, for prothrombin levels to return to normal during the course of therapy in all the cases studied, suggests that hypoprothrombinemia as a cause of serious hemorrhage in salicylate treated patients is an unlikely occurrence. However, should operation be contemplated in such patients the added risk of hemorrhage warrants the use of vitamin K.

The mechanism by which salicylates reduce the prothrombin content of the blood is not clear. Link suggested that a parallelism exists between the effect of dicoumarol and that of salicylates.[10] Doubt is cast upon this relationship by the transient character of the prothrombinopenia in our cases during the continued administration of salicylates, as well as by the suggestion of development of tolerance to the drug indicated by the general trend toward normal prothrombin levels after the third week of treatment. These observations, together with our failure to demonstrate evidence of liver damage, argue against toxic action of salicylates on the liver with resultant prothrombin depression.[18] Further studies of the metabolism of salicylic acid will be required before the precise mechanism of its effect on prothrombin can be determined.

Summary

1. Hypoprothrombinemia of varying degree occurred in 25 cases of acute rheumatic fever treated with massive doses of sodium salicylate.

2. In five cases, bleeding, consisting of epistaxis or small nail bed hemorrhage, occurred at the time of maximum prothrombin depression.

Addendum

Since this paper was submitted, Clausen and Jager[19] have reported a case in which bleeding from the nose and gums occurred in the presence of hypoprothrombinemia induced by salicylates.

Their observation that spontaneous hemorrhage due to the prothrombinopenic effect of salicylates is rare and, when present, is apparently not a factor in causing death from salicylate intoxication, bears out our opinion.

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PLASMOCHIN TOXICITY: ANALYSIS OF 258 CASES *

By Maurice Hardgrove, Col., M.C., A.U.S., and Irving L. Applebaum, Lt. Col., MC., A.U.S., F.A.C.P.

Plasmochin has had a checkered career in the rôle of an anti-malarial agent since it was first introduced into clinical medicine by Mühlens [1] in 1926. The literature is abundant with references which approximate 400 monographs and articles, but it is not within the scope of this paper to present a historical review. Although plasmochin was initially given a trial as the sole anti-malarial drug in various types of the disease, its usage gradually became limited. It has been employed for primary attacks of malaria in combination with quinine and atabrine; in blackwater fever when sensitivity to quinine was suspected; in pregnancy complicated by malaria in order to avoid the oxytocic effects of quinine; in chronic cases in order to curb the recurrence rate; in the follow-up treatment of malaria, when sexual forms were present in the peripheral blood; and in the field for the purpose of mass suppressive treatment. In relationship to control Clark and Komp [2] have reported the results of a 10 year survey in Panama concerning the administration of quinine, atabrine and plasmochin without anti-mosquito measures. Throughout these clinical reports and studies, one is able to note a variable percentage of toxic effects.

Goodman and Gilman [3] have briefly summarized the toxicity of plasmochin in their textbook of therapy. Soon after the initiation of the administration of this synthetic drug for malaria, reports on the poisonous effects were submitted. In 1927 case reports were published by Eiselsberg [4] and subsequently similar observations were made by Namikawa [5] in 1928 and Reyes [6] in 1929. Gastrointestinal symptoms have been noted rather early in the course of drug intake by Clark and Komp,[7] and others. Among those who placed emphasis on cyanosis as an early toxic manifestation were Orachowatz,[8] Chopra and Sen [9] and Manson-Bahr.[10] The question of the pigment causing the cyanotic tinge was in a controversial state. Foy and Kondi [11, 12] have done extensive work on this problem and concluded that methemoglobin rather than pseudo-methemoglobin was the responsible agent. The hemolytic effects of the drug have been noted by Sein,[13] Manai [14] and others. The deleterious effects of plasmochin on the circulatory system have also been recorded,[15, 16] and electrocardiographic studies were performed by Kawahigashi [17] in 1938. In 1943 Slatineanu and Sibi [18] presented the results of investigation of functional tests of the liver and kidney before and after atabrine therapy combined with plasmochin. Among the fatalities published were those by Decherd [19] and Blackie.[20]

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From the Medical Service of Gorgas Hospital, Panama Canal Zone at the time Brig. Gen. Henry C. Dooling, U. S. A. was in charge. Field statistical data furnished by Col. W. C. Cox, M.C.; Lt. Col. S. J. Lerro, M.C.; and Capt. M. Vaughn, M.C.

An acute medical emergency arose as the result of plasmochin poisoning, which gave us an opportunity to study its clinical manifestations. During the month of May 1943, 401 patients were admitted to Gorgas and Margarita Hospitals, located in the Panama Canal Zone, for suspected plasmochin poisoning; 385 of this number were discharged with a substantiated diagnosis. The total number of persons who had received the drug was 4,361. Of this entire group 8.12 per cent (401) were hospitalized, another 2.01 per cent (88) showed mild reactions and were confined to quarters; so that 10.13 per cent exhibited some toxic symptoms.

The course of medicine which these individuals had received for suppressive therapy was as follows: One tablet (0.1 gm.) of atabrine was administered three times daily for five consecutive days. No medication was given on the sixth and seventh days and then for the next five days 0.01 gm. of plasmochin hydrochloride was prescribed three times daily.

The 271 cases admitted to Gorgas Hospital will be described in detail and a comparison will be made with the smaller number entering Margarita Hospital. Of the patients in Gorgas Hospital, 258 (95.2 per cent) were considered as exhibiting evidence of plasmochin toxicity.

All the patients were employed as laborers by the Panama Engineering Division and were ambulatory during the routine course of therapy. They were chiefly black and foreign white male adults in the third and fourth decades of life. No difference in reaction was noticed in the two groups. None of these patients had been immunized against yellow fever.[21] The majority received a total of 15 tablets of atabrine (1.5 gm.) and 13 tablets of plasmochin (0.13 gm.) with a variation of the latter between six and 14 tablets. Twenty-one cases received a total of less than 10 tablets (0.10 gm.) of plasmochin, and of these, seven showed mild signs of toxicity; 12 had a moderate degree and two had severe types of reactions. The majority of the cases were admitted within a 48 hour period (May 14–15, 1943), corresponding to the last day of plasmochin intake and the day following (see table 1).

TABLE I

Relationship of Admissions to Drug Intake

Days	1–5	6–7	8	9	10	11	12	13	14	15	After
Drug—gm.	Total—1.5	0	0.03	0.03	0.03	0.03	0.03	0	0	0	0
No. of hosp. admissions	0	0	0	0	0	8	53	145	20	24	21
	Atabrine	No med.	Plasmochin					No medication			

No definite relationship between past illnesses and the incidence or the degree of toxicity could be ascertained. A past history of malaria was obtained in 92 cases (35.65 per cent), but it did not seem to be a factor in

these cases. It contributed to the splenomegaly which was detected in many cases (24.42 per cent). No history of previous plasmochin therapy was obtained.

Symptoms

The complaints following the oral administration of plasmochin, which were related to its toxicity, were in the order of frequency: Abdominal pain, dark urine (brown, red, black), anorexia, jaundice, headache, nausea, vomiting, feverishness, weakness, malaise, and backache (see table 2).

Table II

Analysis of Symptoms—Plasmochin Toxicity

Symptoms	Cases		Severity			Average Days After Initial Dose	
	Number	%	Mild	Mod.	Severe	Onset	Duration
Abdominal distress	178	68.99	88	71	19	4.49	3.61
Dark urine	144	55.81	42	54	48	3.88	3.99
Anorexia	116	44.96		116		4.40	4.13
Jaundice	115	44.57	48	40	27	4.15	5.40
Headache	100	38.76	58	37	5	4.75	3.37
Nausea and vomiting	87	33.73	41	31	15	4.65	2.28
Fever	64	24.81	Inestimable as a subjective sign				
Weakness, malaise	58	22.48	Undetermined			Undetermined	
Backache	57	22.09	36	18	3	3.86	4.38

Less common complaints were vertigo (7.36 per cent), chest pain (5.04 per cent), diarrhea (3.87 per cent), chills (3.49 per cent), nasal congestion (2.71 per cent), cyanosis (2.33 per cent), photophobia (2.33 per cent), dysuria (1.55 per cent), palpitation (1.16 per cent), prostration (1.16 per cent), syncope (0.77 per cent), and anuria (0.77 per cent). Cyanosis was noticed by six patients but twice this number showed it upon physical examination. Likewise, the incidence of yellow sclerae noticed by the patients and their friends was less than by the examining physicians. In general, it may be stated that the onset occurred after four days of drug administration (total dosage of 0.12 gm. of plasmochin) and that the subjective symptoms lasted from three to four days. This short duration of symptoms was undoubtedly influenced by the immediate treatment the patients received.

Physical Findings

The chief objective findings in the patients admitted were jaundice, general and upper abdominal tenderness, enlarged spleen and liver, cyanosis and pallor. Splenic enlargement, in many cases, was probably due to previous attacks of malaria. Most findings were of slight to a moderate degree and were detectable for a period of from two to five days (see table 3).

About one-half (56.2 per cent) of the patients had a low grade fever, which occurred early during the hospital stay and lasted but a few days. The

TABLE III

Physical Findings—Plasmochin Toxicity

Signs	Cases		Degree			Duration—Days		
	No. 258	%	Slight	Mod.	Severe	Short	Long	Aver-age
Jaundice	138	53.49	58	50	30	1	12	4.5
Rt. U.Q. tenderness	65	25.19	37	21	7	1	10	3.5
Splenomegaly	63	24.42	43	18	2	Inestimable		
Left U.Q. tenderness	59	22.87	28	21	10	1	8	3
Gen. abd. tenderness	54	20.93	28	23	3	1	9	2.5
Hepatomegaly	46	17.82	40	5	1	Inestimable		
Cyanosis	34	13.17	28	5	1	1	6	2
Costo-vertebral tenderness	25	9.69	Undetermined			Undetermined		
Peri-umbilical tenderness	23	8.91	19	4	0	1	8	3.5
Mydriasis	8	3.10	Undetermined			Undetermined		
Shock	7	2.71	1	2	4	1	5	2.5
Nasal congestion	7	2.71	—	—	—	—	—	—
Basal râles	6	2.33	—	—	—	—	—	—
Tachycardia	5	1.94	—	—	—	—	—	—
Epigastric tenderness	2	0.77	—	—	—	—	—	—
Generalized rash	1	0.38	—	—	—	—	—	—

cases considered as seriously ill were lying in bed looking pale and cyanotic, were vomiting and complaining of abdominal distress. They also had yellow sclerae and frequently a lowered blood pressure.

LABORATORY DATA

The urine, which was collected in large bottles alongside each patient's bed, was dark in one-half of the patients on admission or during the hospital stay. The factors in producing darkness were an increase in bile pigment, the presence of oxyhemoglobin and methemoglobin and urinary concentration. About a third of the patients had urine of a red or brown-black appearance, and this served as an index of the severity of the reaction. The urine was acid on admission but after the patients had received large amounts of fluid and sodium bicarbonate, it became alkaline. No true anuria was seen after hospital admission. Renal irritation was noted in less than half of the cases and was manifested by albuminuria, hematuria or casts. Pus appeared in about a third of the cases and marked sediment and hemoglobinuria in less. Most of the abnormal urinary findings disappeared in one to three days which corresponded with the clearing of the rest of the clinical picture (see table 4). Additional laboratory studies and renal functional tests indicated no permanent renal damage.

The hemolytic anemia varied in severity and could be correlated with the intensity of the urinary findings, the elevation of the icteric index and the clinical pattern. Three-fourths of the patients had a red blood cell count below four million and about half (46.5 per cent) had a hemoglobin below 70 per cent. Fifty-six (21.7 per cent) of the cases had an erythrocyte count below two million. Many of the low counts occurred during the acute

TABLE IV
Urinary Findings—Plasmochin Toxicity

	Cases—258		Duration—Days		
	Number	%	Short	Long	Average
Dark urine	129	50	—	—	—
Red or brown-black	86	33	1	7	3
Reaction acid on admission	258	100	—	—	—
Albuminuria	104	40.31	1	7	2
Pus cells	90	34.88	—	—	2–3
Hemoglobinuria	72	27.71	—	—	1–3
Casts	52	20.15	Data not available		
Red blood cells	43	16.28	1	4	1–2
Sediment	35	13.57	—	—	1–2
Urobilin	32	12.4	Corresponded to hemolysis		

reaction after the patient was in the hospital. One red blood cell count dropped precipitously to 800,000. Likewise many (15.8 per cent) of the patients developed a hemoglobin as low as 40 per cent; the lowest was 30 per cent. The mild anemias improved in from three to four days, but the more severe lasted from 10 days to two weeks. Sternal marrow puncture in five patients showed normal regenerating normoblastic bone marrow.

A mild polymorphonuclear leukocytosis (9,000 to 12,000 per cu. mm.) occurred in most patients, though in several the white blood cell count rose as high as 20,000. No increase in lymphocytes was noted. About a fifth (22.48 per cent) of the cases showed an icteric index of over 15 units; one was as high as 175. The average duration of the elevated icteric indices was only three days. The van den Bergh test was indirect in all cases in which the icteric index was above normal.

In a limited number of the more severe cases additional laboratory data were gathered. Blood chemical tests, including glucose, cholesterol-cholesterol ester and creatinine were within normal range in eight cases. Of 162 individual studies of the blood non-protein nitrogen, elevation was noted in 12 cases. These transient rises were observed in patients exhibiting the greatest degrees of jaundice. Red blood cell fragility, Rumpel-Leeds and Donath-Landsteiner tests were normal in six cases. Kidney function tests (phenolsulfonphthalein and urea clearance) were employed in 13 cases; three were slightly reduced, but this was transitory. The renal function, as determined on the basis of the intake and output of fluids, was normal in all cases. During the peak of the illness, liver function studies (hippuric acid test) indicated some impairment in four of the 10 critically ill patients. A return to normal figures was observed after the clinical picture cleared. Of six electrocardiograms, only one revealed a transient abnormality—slight inversion of T_2 and T_3 waves. Spectroscopic examinations of the blood and urine were performed rather late in the course of the disease and therefore results cannot be considered valid. Absorption bands, indicative of methe-

moglobin and oxyhemoglobin, were detected in the urine of three of 15 cases studied, and in the blood of one of 10 cases. However, it may be assumed that methemoglobin accounted for the marked cyanosis and black urinary tint in this series.

Associated findings unrelated to the toxic syndrome were disclosed by laboratory examinations as follows: Helminthiasis 3 cases; estivo-autumnal malaria, 2 cases; latent syphilis 25 cases (serologically); infestation with filaria ozzardi, 1 case; and atypical pneumonia, 1 case.

The correlation of laboratory findings with the clinical picture was evident and the dark urine (urobilinuria, methemoglobinuria and hemoglobinuria), anemia, elevated icterus index, indirect van den Bergh ran parallel with the severity of the toxic process. The laboratory pattern, its transitory nature and response, were typical of an acute hemolytic process due to a toxic agent. There was no evidence of liver, kidney or bone marrow damage of a permanent nature as based on laboratory findings. Temporary renal irritation was expressed by the urinary output of albumin, casts and red blood cells in a number of specimens.

Treatment and Results

A therapeutic regime was immediately established for the treatment of all patients. Diagnostic procedures, which included red cell count, hemoglobin determination and urine analysis upon admission, were employed to guide special therapeutic measures. The routine treatment for each case consisted of the oral administration of thiamine chloride (15 mg. daily), ferrous sulfate (15 gr. daily), vitamin K (2,000 units daily) and sodium bicarbonate (80 gr. daily). Fluids were forced and additional sugar and fruit juices were prescribed for the first few days. Except for mild cases each patient also received 1,000 c.c. of 10 per cent glucose intravenously. If the hemoglobin was below 50 per cent (Sahli) and the red blood cells below 2.5 million or signs of impending shock were present, patients received blood transfusions, which were repeated according to their needs. The rapid influx of patients made it impossible to carry out all orders in extreme detail and the more seriously ill received the most adequate care. Some patients (27) received more than 3,000 c.c. of fluids intravenously and one received 11,000 c.c. in a five day period. Sixty patients received 92 blood transfusions as follows: 37 cases, one transfusion; 15 cases, two transfusions; 7 cases, three transfusions; 1 case, four transfusions.

Blood transfusion was the treatment par excellence for the severe cases of plasmochin intoxication. Its effect was dramatic and it proved to be a life-saving measure in many cases. Even though improvement was shown there was no hesitation in repeating it. Intravenous fluids favorably influenced the clinical course of all patients and were considered an essential phase of therapy. Alkalinization of the urine was used as in black water fever which this syndrome resembles. It aids in preventing the precipitation

of hematin crystals in the renal tubules. Mild cases responded well on the ordinary routine regime.

Of the 271 cases admitted 13 were considered as exhibiting no signs of intoxication; of the rest, 136 were mild; 63 moderate; and 59 severe. The hospital stay averaged 9.76 days for all patients, although some were discharged in three days and others, severe in nature, remained for three weeks. No deaths occurred and no complications or sequelae appeared. The treatment was considered successful in each case.

The 130 patients who were admitted to Margarita Hospital, Margarita, Canal Zone, were similar to the cases we have described above in detail. One hundred and twenty-seven were diagnosed as plasmochin poisoning and 50 of this group showed hemoglobinuria. Eight patients received a total of 12 transfusions and none died. Three cases were excluded because there was no evidence of plasmochin intoxication.

Several illustrative case reports are briefly presented:

Case Reports

Case 1. A 43 year old male, mestizo, laborer, employee of the Panama Canal was admitted to Gorgas Hospital on May 16, 1943 after taking the full course of suppressive treatment as outlined (0.15 gm. plasmochin). Three days prior to hospitalization he complained of abdominal distress, malaise, feeling of slight feverishness and anorexia. On the day of admission he also noted that his urine was darker than usual. Positive objective signs consisted of a slight icteric tinge of sclerae, slight cyanosis of lips and deep tenderness in the right upper quadrant.

Several daily urine specimens were negative except the first specimen which was moderately positive for urobilinogen. Daily complete blood counts were within normal limits. The icteric index on the fourth day was 15, and the van den Bergh test was indirect.

During his hospital stay he was relatively afebrile, but his pulse was slightly rapid for the first three days (90 to 100). He was placed on the routine treatment and in addition received 1,000 c.c. of 5 per cent glucose in normal saline on the first day. The rest of his course was uneventful and he was discharged as well at the end of the week. This is an example of mild plasmochin intoxication with a minimal hemolytic process, responding to a conservative regime.

Case 2: A 25 year old male, mestizo, laborer, employee of the Panama Canal was admitted to Gorgas Hospital on May 15, 1943 after an intake of 0.13 gram of plasmochin during suppressive treatment. Three days prior to hospitalization he complained of headache, nausea, feverishness, jaundice and the voiding of dark red urine. These symptoms persisted to the time of admission.

On physical examination icterus, mild cyanosis and a slightly enlarged tender liver were noted. The first specimen voided was dark red. Blood studies revealed an anemia ranging for the first few days from 1.77 to 2.66 million red blood cells with an average hemoglobin of about 60 per cent. His urine specimens revealed hemoglobinuria and urobilinuria for several days. On the fourth hospital day his icteric index was 15, non-protein nitrogen 26.9 and the van den Bergh reaction was delayed indirect.

The patient ran a low grade febrile course for the first few days. He was placed on routine therapy and in addition received a 500 c.c. blood transfusion on the second day and also 1,000 c.c. of 5 per cent glucose in normal saline intravenously. Gradual improvement was noted and he was discharged as well on the tenth day. This is an

example of a moderately severe case of plasmochin intoxication with a satisfactory response to routine therapy and a blood transfusion.

Case 3. A 35 year old, black, male laborer, employee of the Panama Canal, was admitted to Gorgas Hospital on May 15, 1943 after an intake of 0.12 gram of plasmochin during suppressive treatment. Two days prior to admission he complained of abdominal distress, malaise, headache, nausea, vomiting, anorexia, jaundice and the voiding of very dark urine.

Physical examination revealed an acutely ill individual in mild shock with a small rapid pulse, exhibiting signs of marked jaundice and moderate cyanosis. General abdominal tenderness was elicited on deep pressure and he had moderate mydriasis.

The red cell count was 1.9 million red cells on admission and for several days was under 2.5 million. The hemoglobin was 50 per cent. The initial white cell count was 18,000 with a polymorphonuclear trend. The urine had a blackish tint on the first day and was dark red on the second day. Albuminuria, methemoglobinuria and hemoglobinuria were detected for several days. Kidney function tests were subnormal (phenolsulfonphthalein was 20 per cent) on fourth day, but on the eighth day the urea clearance was normal. Blood examinations on fourth day revealed an elevated non-protein nitrogen (44.6 mg.) and normal blood sugar (80 mg.). The initial icteric index was 42 units. A sternal puncture indicated an active bone marrow response to hemolysis. Spectroscopic examination of the blood revealed the presence of methemoglobin. Electrocardiographic studies showed slight inversion of T_2 and T_3 waves.

The patient had a temperature of 100 to 102° F. during the first week and was seriously ill. During this period he received three blood transfusions and three intravenous infusions of 5 per cent glucose in normal saline. He gradually felt better, became afebrile, the urine analysis reverted to normal and his red cell count rose to 3.15 million and 72 per cent hemoglobin. He was discharged at the end of two weeks. This is an example of a case of severe plasmochin intoxication with severe hemolysis and methemoglobinuria and the rare findings of shock, mydriasis, renal irritation and toxic myocarditis (see figure 1). Energetic measures were necessary in the therapy of this case.

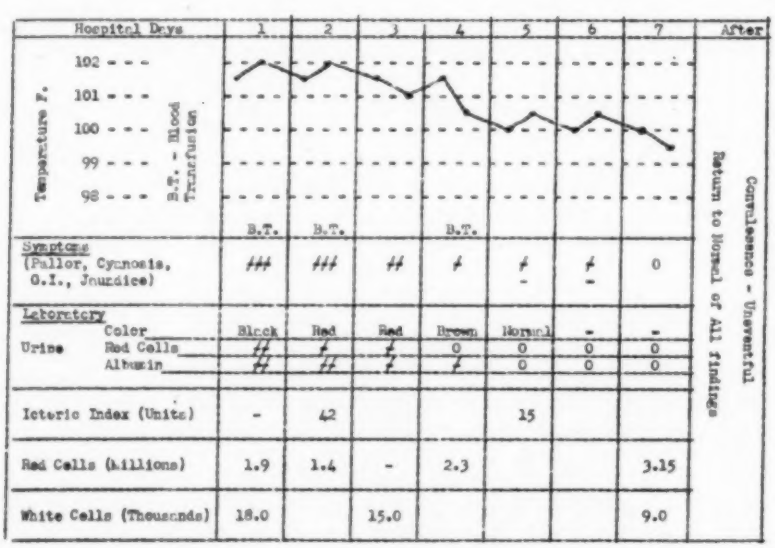

FIG. 1. Composite chart of case 3 (severe plasmochin intoxication).

Comment

On the basis of the data in this report, it is evident that plasmochin is a dangerous drug and due caution must be exercised in its administration. It has a limited use and at present it is employed at Gorgas Hospital in selected recurrent cases of malaria and all cases which persistently reveal the sexual forms of the parasite in the peripheral blood, after an adequate course of another anti-malarial drug (atabrine or quinine) has been prescribed. It is recommended that the dosage of plasmochin be modified because of the small margin of safety and the individual sensitivity occasionally encountered. At present the dosage at Gorgas Hospital has been reduced to 0.01 gm. twice daily for a period of three days. It is also felt that plasmochin should not be used in the field, but as a rule in a hospital, where more careful observation by the physician is possible. When early signs of toxicity appear, the drug must be discontinued. If jaundice, pallor or impending shock occur, then energetic measures of therapy as outlined (blood transfusion, intravenous fluids, etc.) should be instituted. Laboratory studies, particularly daily blood counts and urine analysis, are important in the diagnosis, treatment and follow-up of each case.

The rôle of atabrine in the introduction or accentuation of toxic symptoms cannot be fully evaluated in this series.

Conclusion

Of 4,361 laborers who were placed on a suppressive ambulatory routine for malaria (five day course of 1.5 gm. atabrine followed by two days of no medication and then a five day course of 0.15 gm. plasmochin hydrochloride), 489 cases (10.13 per cent) developed toxic manifestations of plasmochin poisoning. The individual syndrome varied in severity but the more seriously ill presented the typical clinical and laboratory pattern of acute intravascular hemolysis. All cases recovered upon a therapeutic regime which included alkalinization of the urine, intravenous fluids and blood transfusions, and no permanent effects were noted. Plasmochin is a drug which presents a potential danger to the patient and therefore the reduction in dosage and the careful observation of patients receiving the drug are recommended.

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INFECTIOUS MONONUCLEOSIS: REPORT OF AN EPIDEMIC IN AN ARMY POST *

PART I

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EPIDEMICS of infectious mononucleosis [1] are not infrequent, especially where groups of young individuals live in close proximity. Outbreaks in colleges and schools have been the classical examples of such epidemics. They have also occurred in military installations. The first epidemic in this country was described by West [1 (k)] in 1896.

The post, in the period during which the epidemic took place, was of diverse and changing composition. In the main, it consisted of the station complement and a large number of battalions in training. The former and smaller component consisted of individuals who usually had had from one to several years of service and its composition changed but slowly. The battalions, on the other hand, arrived and departed at irregular intervals, varied in strength between 700 and 1000 men, were mostly recent inductees and remained on the post usually for a period of four to six months.

Before embarking on a description of the epidemic, the diagnostic criteria we used must be stated. This is necessary not only because of the protean manifestations of the disease and its unknown etiology, but because of the large number of cases with insidious onset and mild symptoms or with no clinical manifestations whatsoever. The latter were admitted for some totally unrelated condition. That there can be many such cases during an epidemic, has been amply confirmed by Halcrow and co-workers [2] in a recent outbreak of the disease in an E.M.S. hospital in Scotland. Of the 298 individuals whom they examined, a group composed of both patients and nursing staff, 97.9 per cent showed evidence of the disease. Only 125 of these were clinical cases, whereas 165 had blood and serological changes but no clinical manifestations. What was even more remarkable was the demonstration of many cases in the adjacent town and a few in two towns situated 13 and 20 miles from the hospital. It is interesting that Baldridge,[3] as early as 1926, asserted that sporadic cases are only the more marked instances of a small epidemic in which many of the cases are so mild as to escape notice.

Both in the absence of clinical manifestations and as confirmation of the suspect clinical syndrome, the diagnosis is based on the presence of "leukocytoid" lymphocytes in the peripheral blood, a mononucleosis, a positive

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heterophile antibody agglutination test, or a combination of these three. The application of these criteria to the large number of cases seen in an epidemic such as we are describing presents certain difficulties.

A number of workers have denied that "leukocytoid" lymphocytes are pathognomonic of infectious mononucleosis. Baldridge et al.[3] found varying percentages of abnormal lymphocytes in many infections associated with lymph node involvement. In a number of cases of upper respiratory infections, such as acute pharyngitis and acute sinusitis, Warren [4] reported that many of them showed from 1 per cent to 10 per cent of cells characteristic of infectious mononucleosis. Fifteen heterophile antibody agglutination tests were performed in these cases and all were negative. He admitted, however, that serial blood counts and heterophile antibody agglutinations were not complete. Randolph and Gibson [5] studied 24 allergic students, nurses and hospital personnel whose major complaint was fatigue or weakness and found atypical lymphocytes present in a range from 2 to 27 per cent. One was known to have had infectious mononucleosis two years earlier and eight had or had had cervical gland enlargement. A heterophile antibody determination was performed in 22 and the highest titer was 1 : 32. In a continuation of these studies in a group of 140 student nurses, Gibson and Hettig [6] reported that atypical lymphocytes varied between none and 22 per cent. On the basis of the single blood count performed, however, the number of such lymphocytes bore no relationship to a past history of allergic disease. Infectious mononucleosis had been previously diagnosed in two because of a positive heterophile antibody determination. This test, unfortunately, was not a part of the study. Although the authors discount the possibility that they were dealing with an epidemic of infectious mononucleosis or with cases in the recovery phase of this disease, no convincing evidence is presented. No Davidsohn absorption tests were performed. The occurrence of seronegative cases and the frequent, rapid disappearance of elevated heterophile titers during convalescence are not taken into consideration. The reported cases were in a group in whom infectious mononucleosis is common and it is known that atypical lymphocytes can persist for months and even for years after an attack. They have been reported as long as 7 and 10 years later.[3, 7] Finally, atypical lymphocytes are apparently common in infectious hepatitis. In 1923, Jones and Minot [8] reported 26 sporadic cases in whom such cells reached 50 per cent during the height of the lymphocytosis. No heterophile antibody determinations were performed. Recently, Barker, Capps and Allen [9] found 5 to 20 per cent and occasionally as high as 60 per cent of these cells in an epidemic of infectious hepatitis. Heterophile antibodies were consistently absent on repeated examinations.

A mononucleosis at some stage of the disease has been considered the essential sign in infectious mononucleosis by Bernstein.[10] He, however, quotes Chevallier to the effect that in some instances the mononucleosis may be so slight that the diagnosis depends only on the presence of abnormal cells. His conclusion, moreover, is based on sporadic cases which are usually severe

enough to warrant prolonged observation and frequent blood counts. In addition, there is a general agreement that the mononucleosis is much higher in sporadic cases than in those seen in an epidemic.[1 (e), 1 (j), 3, 11]

Although the Paul-Bunnell test [12] has been a great advance in the diagnosis of infectious mononucleosis, it has many limitations when applied to the epidemic disease. The variability in the time of appearance of the maximum titer and the occasional rapid disappearance of elevated values [10, 13, 14, 15] are great disadvantages where repeated tests are not feasible or where large numbers of asymptomatic cases are concerned. The number of sero-negative cases is not inconsiderable. In the sporadic form, the percentage of positive tests has varied between 43 per cent and 100 per cent.[13] The variation has depended to some extent upon the frequency with which the test has been performed in the individual cases, the technic employed and the titer that was considered as a positive test. More important, however, has been the number of cases the author has excluded merely because the test was negative.[13, 16, 17] Such a procedure has even been recommended.[18] On the other hand, the test is not specific as not only have rare false-positives been reported [19, 20, 21] but normal sera can and often do contain sheep-cell agglutinins. Excluding serum sickness and converting the values into final dilutions, the maximum titers that have been reported in the literature are shown in table 1. Although titers above 1 : 64 and 1 : 80 occur in only a very small number of controls, the fact that they do occur is more important than the actual percentage where clinical manifestations are completely absent or mimic a great variety of other illnesses. The Davidsohn absorption test [30] is undoubtedly of greater usefulness under such circumstances. This test, however, is more complicated and places a heavy burden upon a laboratory staff already overtaxed with other duties, particularly when large numbers of patients are involved. The introduction of modifications in technic which shorten the test [13, 31] do not decrease its complexity. Moreover, the absorption test as performed in our laboratory did not give uniform results. This was true of both the guinea-pig and the beef red cell suspensions and will be more fully discussed later.

TABLE I

Maximum Titer of Sheep-Cell Agglutinins in Control Series

Author	Number of cases tested	Maximum titers	Author	Number of cases tested	Maximum titers
Barrett [16]	100	1 : 20	Bunnell [26]	2015	1 : 128
Davidsohn [22]	217	1 : 56	Butt and Foord [27]	436	1 : 128
Smeall [23]	765	1 : 64	Stuart [28]	300	1 : 320
Shaw and Macgregor [24]	136	1 : 64	Beeukes [21]	100	1 : 516
Bernstein [25]	300	1 : 80	Demanche [29]	147	1 : 896

In all suspected cases in this epidemic, several blood counts and at least one heterophile antibody agglutination test were performed, but no systematic attempt was made to determine the maximum percentage of "leuko-

cytoid" lymphocytes, the maximum mononucleosis or the highest heterophile antibody titer attainable. Davidsohn absorption tests were done in a number of typical cases throughout the period of study merely to confirm the fact that we were dealing with an epidemic of infectious mononucleosis. They were also performed in doubtful cases and in those who presented a German measles or scarlatiniform rash, jaundice or pneumonia. More extensive studies were undertaken only in those patients who required prolonged hospitalization, either because of their admitting illness or because of the severity of the infectious mononucleosis. The ordinary cases with clinical manifestations of the disease were usually symptom-free after four to five days of hospitalization and anxious to return to duty. It was not deemed advisable to interfere with their training program solely for the purpose of making further blood studies.

For the purposes of this report, we have arbitrarily excluded all cases who had less than 10 per cent "leukocytoid" lymphocytes of the total white blood cell count, unless the heterophile antibody agglutination titer was 1 : 224 or above or was positive with the Davidsohn test. We realize that many cases of infectious mononucleosis were excluded by these criteria, either because of insufficient blood studies or because they were admitted to the hospital in a late convalescent stage. During the height of the epidemic, practically every patient in the hospital showed varying percentages of abnormal lymphocytes in their blood smears. As only those cases actually seen at the hospital form the basis of this report, it is evident that there must have been large numbers without clinical manifestations or with such mild symptoms that they were never hospitalized. Many of the latter either stayed on duty or were confined to quarters for a day or two.

This study includes only the 556 cases admitted or examined at the hospital between January 1, 1943, and February 29, 1944. Up to July, 1944 an additional 131 cases were admitted but have not been tabulated, as the essential facts and conclusions would not have been altered by their inclusion.

Distribution

A. Monthly Incidence. The outbreak began in August 1943, but sporadic cases were recognized prior to this date. In the preceding seven months there were 11 cases diagnosed as infectious mononucleosis.

The monthly incidence is shown in table 2. It will be noted that the admission rate rose rapidly to its peak in October and November 1943 and

Table II
Distribution by Month of Admission

Month	Number of cases	Month	Number of cases
Jan. to Aug. 1943	11	November 1943	147
August 1943	18	December 1943	104
September 1943	46	January 1944	59
October 1943	139	February 1944	34

then declined more slowly. Small numbers of cases were admitted in the succeeding months of 1944 not covered by this report. The numbers in these months were as follows: March, 40; April, 34; May, 39; June, 18.

Most epidemics have occurred in the spring and fall.[10] However, the epidemic described by Halcrow et al.[2] in England, which had many similarities to our own, began in August. Although sporadic cases are more frequent in the winter months, no month is spared.[10, 32, 33] June is the hottest month of the year in this region and it stays warm until October. Although the height of the epidemic was in the fall, it began in a very hot August and cases were admitted as late as the following June.

B. Age. The cases which we are reporting consist of military personnel exclusively and therefore belong to a limited age group. The distribution by age of the epidemic cases and of the army as a whole are shown in table 3. The latter is a compilation based on personnel surveys as of December 31, 1943 and June 30, 1944.

TABLE III

Age Distribution of Epidemic Cases and of Army as of December 31, 1943 and June 30, 1944

	Epidemic Cases		Army December 31, 1943	Army June 30, 1944
Age	Number of cases	Percentage	Percentage	Percentage
18	57	10.2	3.6	0.7
19	59	10.6	6.9	4.0
20	58	10.4	7.6	7.1
21	35	6.2	8.4	7.3
22	37	6.6	8.7	7.8
23	34	6.1	7.9	8.4
24	34	6.1	7.7	7.8
25	31	5.5	7.1	7.5
26	33	5.9	5.4	7.0
27	15	2.6	5.0	5.7
28	22	3.9	4.4	5.0
29	22	3.9	4.0	4.4
30	18	3.2	3.4	4.0
31	19	3.4	3.1	3.5
32	19	3.4	2.6	3.0
33	13	2.3	2.4	2.7
34	13	2.3	2.1	2.4
35	12	2.1	2.0	2.2
36	7	1.2	1.7	2.0
37	8	1.4	1.5	1.8
38	8	1.4	1.0	1.5
39	0			
40	1			
41	0			
42	1			
43	1	0.7	2.5	2.1
44	0			
45	0			
46	0			
47	1			

The ages of the patients ranged between 18 and 47 years. From the table, it is evident that there was a greater frequency of the disease at age

18 to 20 years inclusive when compared to their percentage strength in the army. This was very marked in the 18 year old group, the frequency being 3 to 14 times as great. From ages 21 to 25 years inclusive, the case rate was somewhat lower and from 26 to 38 years inclusive, it closely approximated the percentage age composition of the army. After 38 years, there was only an occasional case and a much lower incidence than the expected rate.

All authors have stressed the fact that infectious mononucleosis is largely a disease of children and young adults.[10] As a classical example of this, the outbreak at the Lawrenceville School [1 (e)] is often cited. Adults, however, are not immune. In an epidemic in the Falkland Islands,[34] 10 per cent of the cases were between 45 and 65 years of age and in the one described by Halcrow et al.,[2] most of the patients were between 20 and 45 years of age. Two patients were aged 64 and 84 years respectively.

Analysis of the age incidence in the epidemic indicates that the younger individuals, especially those 18 years of age, are more susceptible to the disease. It also indicates that the so-called relative immunity of adults, at least until age 39 years is reached, does not obtain where a group lives closely together and where the opportunities for contact are favorable.

C. Sex. There were four females in this series, or 0.7 per cent of the total number. This is proportional to their representation on the post, as hospital admissions were limited to nurses and WACs, and did not include dependents.

D. Color. Infectious mononucleosis in the negro is supposed to be a rarity. Bernstein,[10] in 1940, could find but a single case [35] reported in the literature up to that time.

Since then, there have been 15 additional cases. In 1941, Werlin, Dolgopol and Stern [36] reported four cases and noted that all were seen in one month and from the same district of New York City. Ray and Cecil,[37] in 1944 reported three cases, one of which was complicated by sickle cell anemia and in the same year, Johnson [38] reported two cases in children. Recently, Blain and Vonder Heide [39] reported six cases, two of which, however, were doubtful by our criteria as no atypical lymphocytes were noted.

There were 49 negroes in this series or 8.7 per cent of the total number of cases. Except for the month of August, 1943, when the outbreak started and during which the case rate was the lowest, this percentage was considerably higher than the percentage colored strength on the post. For the last five months of 1943 and the first two months of 1944, the period covered by this report, the percentage colored strength was as follows: August, 10.2 per cent; September, 3.6 per cent; October, 3.2 per cent; November, 5.8 per cent; December, 5.0 per cent; January, 3.2 per cent; February, 2.9 per cent. The average for these seven months was 4.8 per cent or about half the actual incidence.

These statistics indicate that infectious mononucleosis is not a rarity among the negro, but that, on the contrary, he apparently has a greater sus-

ceptibility to the disease. They support Bernstein's [10] contention that the paucity of reported cases in the negro is due to the fact that members of this race with minor illnesses are not hospitalized.

E. Race. We have encountered the disease in all races including those of Hindu and Chinese extraction. Italian prisoners of war were represented by 11 cases. There were 24 Jews in this series or 4.3 per cent of the total. It was our impression that this percentage was also approximately their strength on the post and that they did not exhibit any increased susceptibility to the disease.

F. Organizations. Every outfit was represented by one or more cases. Twenty-one organizations contributed 10 or more cases. The Station Hospital personnel with 35 cases was the largest group.

Bernstein [10] is of the opinion that the frequency of infectious mononucleosis among individuals working about a hospital has been overemphasized. He feels that these individuals are promptly hospitalized for the treatment of even trifling illnesses and are, therefore, correctly diagnosed.

This situation, however, did not obtain in the post. Minor illnesses among line troops were more frequently hospitalized than among service troops. Because of the arduous physical exertion required of the former, the unit medical officer recommended hospitalization in the vast majority of cases if there was a significant temperature elevation.

We must also emphasize the fact that no epidemiological survey of the hospital personnel was undertaken and that the discovery of the disease depended upon the same factors as in the other troops. It would, therefore, favor the belief that the epidemic disease is actually more frequent in hospital personnel, presumably due to the increased opportunity of contact with persons afflicted with the disease.

Clinical Picture

1. Clinical Types. In a disease as protean in its manifestations as infectious mononucleosis, we have found it both interesting and instructive to classify the cases in this epidemic into clinical types. This, at times, was not a simple matter as many cases did not readily fit into any arbitrary classification. In large numbers, manifestations of several types were present either simultaneously or at various times during the course of the illness.

We have classified the cases on the basis of the outstanding clinical feature, such as the presence of jaundice, skin eruptions, meningitic signs, pneumonia, etc. The importance of such a classification in the differential diagnosis is apparent. In table 4 are listed the various clinical syndromes we have encountered and their frequency.

2. Mode of Onset. In contrast to sporadic cases, the onset in the majority was acute, with a chill or chilly feelings and fever. In a few of the anginose, but especially in the icteric and lymphoglandular types, there was a period varying from several days to two weeks, in which there were vague constitutional symptoms such as malaise, fatigability, headache and anorexia,

TABLE IV
Distribution by Clinical Types

Type	Number of cases	Percentage	Type	Number of cases	Percentage
Anginose	256	47	Pulmonary	30	5
Insidious	112	20	Abdominal	12	2
Eruptive	92	16	Lympho-glandular	11	2
Icteric	34	6	Meningitic	9	2

with or without a mild sore throat. The insidious cases were discovered by routine blood count in soldiers admitted to the hospital for a variety of other conditions. Even in the acute cases, the date of admission was rarely the day of onset of the disease, as most of the soldiers attempted to remain on duty without going on sick call or were treated in the dispensaries by their unit surgeons. Some were referred to the Skin Clinic because of the discovery of an eruption, while others developed the disease while under observation in the hospital. In two cases, the finding of a positive blood Kahn reaction led to other blood studies.

3. Initial Symptoms. From the classification into types, the initial symptoms and their frequency can be inferred. In the anginose type, the patients complained of chills or chilly sensations, fever, sore throat, malaise, headache, generalized aches and pains, sweats, and anorexia. Depending upon the severity of the sore throat and upon the extent of involvement of the respiratory tract, a non-productive cough, coryza, pains in the anterior part of the chest, painful deglutition and hoarseness were not infrequent. Dizziness, nausea, and vomiting occurred but were not common. A few cases were ushered in by an epistaxis.

The pneumonic type was merely a variant of the anginose type, differing from it in the severity of the cough and the presence of pain in the chest. When this syndrome was accompanied by a polymorphous eruption, the patient appeared seriously ill.

Although the insidious type had no clinical manifestations of the disease and the patients were admitted for unrelated conditions, careful questioning frequently elicited a history of a mild sore throat one or more weeks previously. A mild cervical or generalized lymphadenopathy was practically always present and at times the spleen was palpable.

In the eruptive form, although the mode of onset was similar to the anginose type, the patient exhibited skin lesions on admission or developed them shortly thereafter. In a few cases the upper respiratory symptoms were mild and the patient sought admission solely because of the eruption.

The initial symptoms in the icteric type were variable, depending on the presence or severity of the throat involvement. Where the sore throat was absent or mild, the symptoms were less violent, the patients entered the hospital at a much later date and the prominent complaints were malaise, anorexia, nausea, vomiting and epigastric distress, followed by jaundice.

Fever was absent or mild. In those with a moderate or severe sore throat, fever, chills and headache were present in addition to the above symptoms.

Upper respiratory symptoms were absent or mild in the abdominal type and the presenting symptoms were pain in the abdomen, nausea and vomiting. The pain was situated in the right lower quadrant, the right upper quadrant or the epigastrium.

In the lymphoglandular type, the sore throat was mild or had disappeared and the patient's only complaint was painful swollen glands. Although the lymphadenopathy was invariably generalized, the most prominent glands and the ones which were the most painful were always the cervical lymph nodes, except in one instance, where the pain was confined to the inguinal region. Lesser pains were not infrequently complained of in the axillae, the groins, or both.

In the meningitic type, the anginose symptoms were moderate or severe but in addition, there were severe frontal headache and pain and stiffness of the neck. These were occasionally accompanied by vomiting.

Upper respiratory symptoms were absent in the malarial form, the complaints being daily chills, fever, headache, malaise and sweats.

4. Duration of Attack. It is very difficult to arrive at any satisfactory conclusion concerning the duration of the attack in the cases in this series. Neither the duration of clinical manifestations nor the duration of fever applied to all patients. The insidious cases exhibited no clinical manifestations at the time of admission. On the other hand, there were 50 cases who presented such varied clinical manifestations as icterus, mild angina, stomatitis or skin eruptions and yet were afebrile on admission. Some of both groups, however, acknowledged a recent, mild sore throat while many stated that they had had chills or fever or both at some time prior to admission. It is of interest that in six of this afebrile group a relapse occurred which was accompanied by fever.

Another factor was the great variation in the period between the onset of the disease and admission to the hospital. This interval ranged from a few hours to 25 days in 417 cases in which the data concerning this period were reliable. The average number of days of illness prior to hospitalization for this entire group was three. In 19 cases the onset of the disease occurred in the hospital while under observation for some other, unrelated condition.

There were 377 cases who exhibited an elevated temperature with the acute phase of the disease. The duration of the febrile course in the hospital ranged from one to 35 days. The elevation lasted for only one day in 97 of these cases. Arranged according to weeks of temperature, the duration was as follows: One week or less, 89 per cent; one to two weeks, 9 per cent; two to three weeks, 1 per cent; three to four weeks, 1 per cent. This is in marked contrast to the sporadic cases reported in the literature.[10] These figures do not include the prolonged low-grade fever in convalescence or the relapses. There were 18 cases who exhibited a low-grade fever for a period

varying between two weeks and three months. As this manifestation was disregarded in judging the soldier's fitness for duty, the actual persistence of this phenomenon cannot be given. Relapses will be discussed elsewhere.

In contrast again to sporadic cases,[10] the peak of the temperature in the vast majority of instances occurred on the first or second day. The temperature peaks in this series are shown in table 5. The insidious cases accounted for the large percentage with a peak of less than 99° F. The highest temperature recorded in this series was 105.6° F.

TABLE V

Peaks of Temperature

Temperature	Percentage of cases	Temperature	Percentage of cases
98° to 99° F.	26	102° to 103° F.	14
99° to 100° F.	15	103° to 104° F.	12
100° to 101° F.	12	104° to 105° F.	6
101° to 102° F.	14	105° to 106° F.	1

5. Pulse. In general, the pulse rate paralleled the temperature. There were a few cases with temperatures over 102° and less frequently with temperatures over 103° whose pulse rates were only 80 to 88. What was especially striking, however, was the great frequency of bradycardia in convalescence.

6. Respiratory System. Subjectively, sore throat was the commonest complaint. Although angina was the prominent objective feature in only 47 per cent of the cases as shown in table 4, it must be emphasized that the majority of those belonging to the other types exhibited evidence of involvement of the pharynx at some time during their hospital stay so that signs of throat involvement were present in 73 per cent of the patients. This percentage would have been much greater, if there were added to it the insidious cases with a history of sore throat and those acute cases whose sore throat had subsided by the time they entered the hospital. In some patients, especially those belonging to the icteric type, the sore throat appeared later in the course of the disease and in still others, throat involvement occurred with a relapse.

There were five types of throat infection: diffuse injection of the pharynx, tonsils or both of varying degrees (68 per cent); follicular tonsillitis or pharyngitis (18 per cent); ulcerative tonsillitis or pharyngitis (9 per cent); membranous tonsillitis or pharyngitis (3 per cent); stomatitis (2 per cent). No case developed a peritonsillar abscess. The stomatitis was of the aphthous variety and was very diffusely distributed, involving the pharynx, tonsils, soft palate, buccal mucous membranes, gums, tongue and even lips. In one confusing case with marked ulcerative stomatitis, a bullous eruption appeared on the palms and soles. Several others were complicated by a polymorphous eruption.

The soft palate was very frequently involved in the various types of throat infection and in a few cases exhibited scattered petechiae. A gingivitis was extremely common. It was usually mild and consisted of puffiness, redness and easy bleeding of the gums. In 52 cases, it was severe and ulcerative in character. Epistaxis was rare, occurring in only four cases. When it did occur, however, it was of moderate severity, ushered in the illness and was the only finding on admission. Herpes labialis was rare, being mentioned in only six cases.

Although cough was a common symptom, pulmonary findings were present in only 30 cases. The initial symptoms did not differ from the anginose type except for the severity of the cough and the more frequent complaint of pain in the chest. The cough was of the spasmodic type and was frequently described as pertussis-like. Audible wheezing was occasionally present. It was only slightly productive of thick, tenacious sputum. In only one case was it blood-tinged. The pain in the chest was usually sternal but occasionally referred to either lung. Numerous diffuse sibilant and sonorous râles, inspiratory and expiratory, were heard without change in the percussion note or breath sounds. In a few cases, fine râles in addition to the rhonchi were audible over a portion of the lung. In 14 of these cases, a pneumonitis was demonstrated by roentgen-ray. The roentgen-rays were not always positive on admission but became so two or three days later and in one instance, they became positive with a relapse on the eighth hospital day following three days of normal temperature.

The highest temperature in this group varied between 99.2° and 105° F., and the febrile course varied between one and three days in all except two cases. In these two, it lasted six days. The latter were the only ones who appeared very ill and were also the patients with the highest temperature. One of these, whose temperature rose to 104.8°, was taken ill, two days before admission, with chilliness, fever, sore throat, cough, malaise, generalized aching, nausea and vomiting. He showed a severe, ulcerative stomatitis, involving pharynx, tonsils, soft palate, buccal mucous membranes and gums. There were pronounced asthmatic wheezing, moderate cyanosis, diffuse, sibilant and sonorous inspiratory and expiratory râles and moderate fine moist râles over the right lung posteriorly. Roentgen-rays revealed a mottled density involving the entire right lung and the first, second and third left interspaces. The day after admission, he developed a generalized polymorphous eruption, papular, nodular and vesicular in character. The other case, whose temperature rose to 105°, had been ill for five days prior to admission with the usual symptoms. Besides a moderate pharyngitis and diffuse rhonchi over both lungs, he also exhibited a generalized polymorphous eruption on entrance into the hospital. His roentgen-ray revealed a diffuse hazing of the upper two-thirds of the right lung except for the apex.

The rest of the cases were all mild and indeed, the finding of a pneumonia and its extent were always a surprise. In one remarkable example, a WAC who had been ill for five days with chilliness, sore throat, fever and

cough, the highest temperature was 99.2°, which continued for three days. The roentgen-rays of the chest showed a pneumonitis involving the entire left lung field except the apex and costophrenic angle. Another, who had been ill for only one day and whose roentgen-ray showed a pneumonitis involving the medial third of the lower right lung field, had no temperature on admission. His highest temperature was 99.2° on the second hospital day.

Some type of angina was present in all. Herpes labialis was present in four cases. The laboratory findings were interesting. The sputum did not contain pneumococci or other significant organisms on typing or culture. Vincent's organisms were found on throat smears. Blood cultures were sterile. The blood counts shortly after admission were variable. The white blood cells varied from 4,600 to 32,000 and the neutrophiles from 48 to 86 per cent. The majority showed a normal or elevated count with an increased number of neutrophiles. The others showed a normal white blood cell count or a leukopenia with a moderate lymphocytosis. In from several days to a week, there was a shift to a leukopenia and a mononucleosis in the first group and an increased mononucleosis in the second. The lowest count in this period was 4,000 and the largest percentage of mononuclears was 67 per cent. This was in keeping with the hematological findings in the anginose group in general.

The roentgenological appearance of the lesions (figures 1, 2, 3, 4, 5) were indistinguishable from those described in atypical pneumonia and consisted of a haziness or mottling through which the ribs could be seen. The right lower lobe alone was involved in five cases, the left lower lobe in two, the right middle lobe in two and the right upper lobe in one case. In three cases, more than one lobe was involved. Clearing of the roentgen-ray findings took place very rapidly.

In the remaining 16 cases, the roentgen-rays were negative in all but two. In one patient, who coughed up bloody mucus, a roentgen-ray shortly after admission revealed a haziness in the left lower lobe suggestive of an early pneumonitis. However, two days later, another roentgen-ray was reported as negative. In the other, the initial roentgen-ray was reported as showing changes in both lower lung fields suggestive of acute bronchial irritation. Another film taken four days later was negative.

The literature contains many references to the similarity between the cough in these cases and that in pertussis and to the likelihood that enlarged intrathoracic glands are the cause in both. We have been unable to demonstrate enlarged mediastinal glands in any of this group or in 23 additional patients in whom chest roentgen-rays were taken because of the severity or character of the cough. Râles were not audible in the latter group.

Whether the acute bronchitis or actual pneumonitis, present in 2.9 and 2.5 per cent of the patients respectively, was a complication due to secondary invaders or part of the disease process itself, cannot be stated with certainty. There were many striking similarities between the type of pneumonitis we

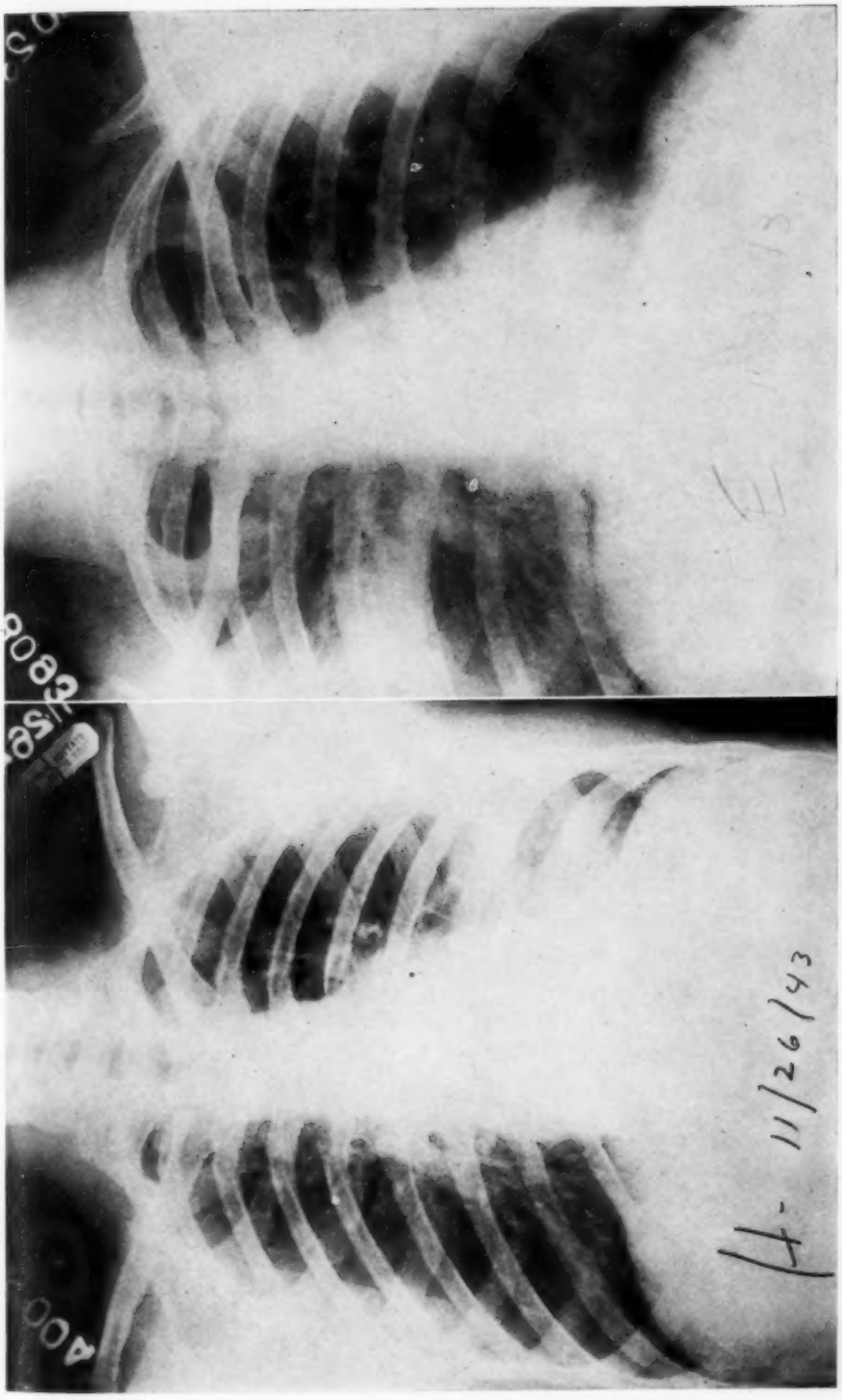

FIG. 1.

Roentgenologic characteristics of the pulmonary involvement.

FIG. 2.

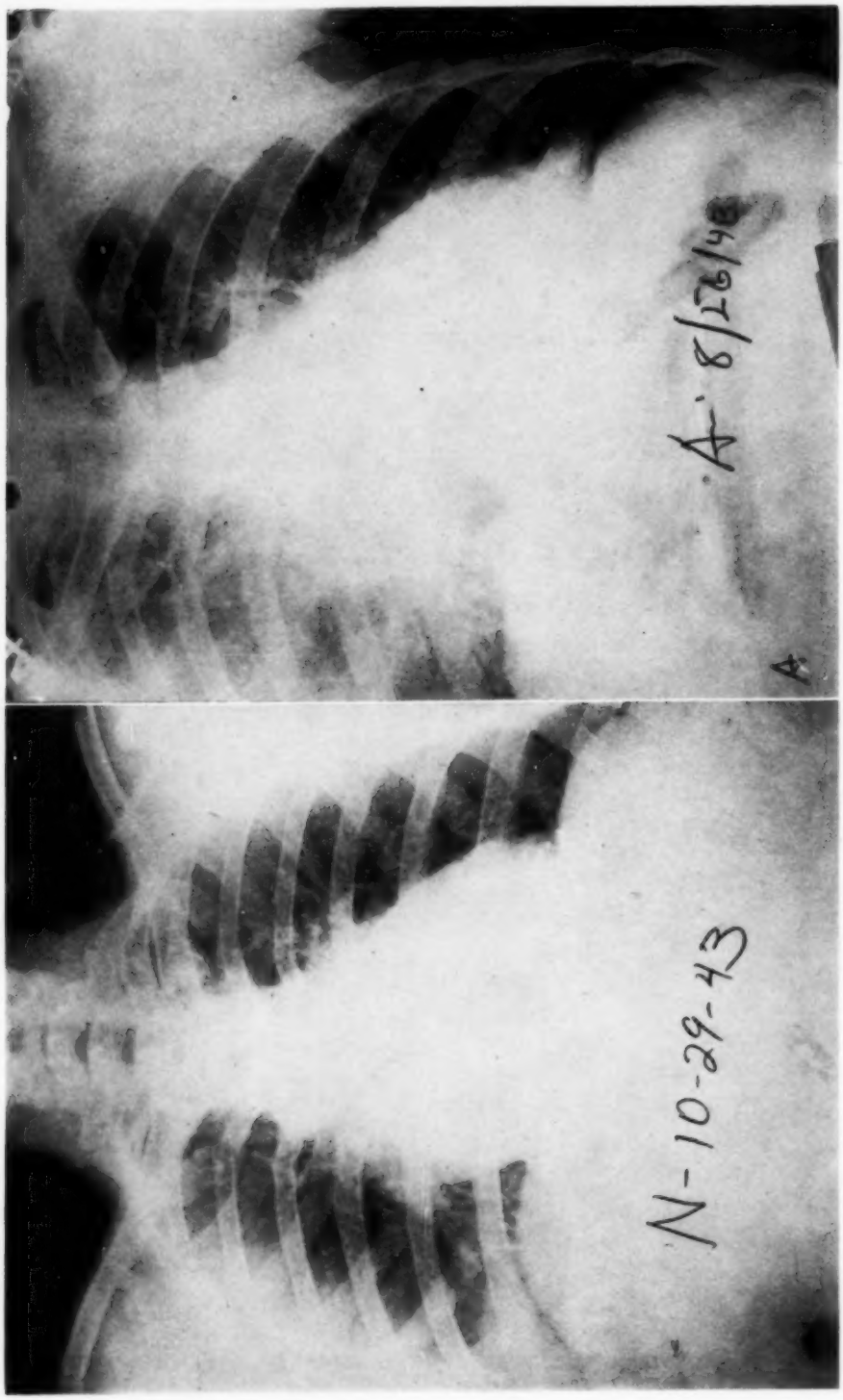

FIG. 3.

FIG. 4.

Roentgenologic characteristics of the pulmonary involvement.

observed and that present in atypical or "virus" pneumonia. Among these were the character of the cough, the asthmatic wheezing, the disparity between the physical signs and the extent of the pulmonary involvement, the roentgenologic picture and failure to respond to sulfadiazine. Recently, increased heterophile antibody titers have been reported in "virus" pneumonia.[40] We have seen three similar cases, one each in October, November and December, 1943. The first patient had an extensive involvement of the right lower lobe and his heterophile agglutination titer rose from 1 : 28 to

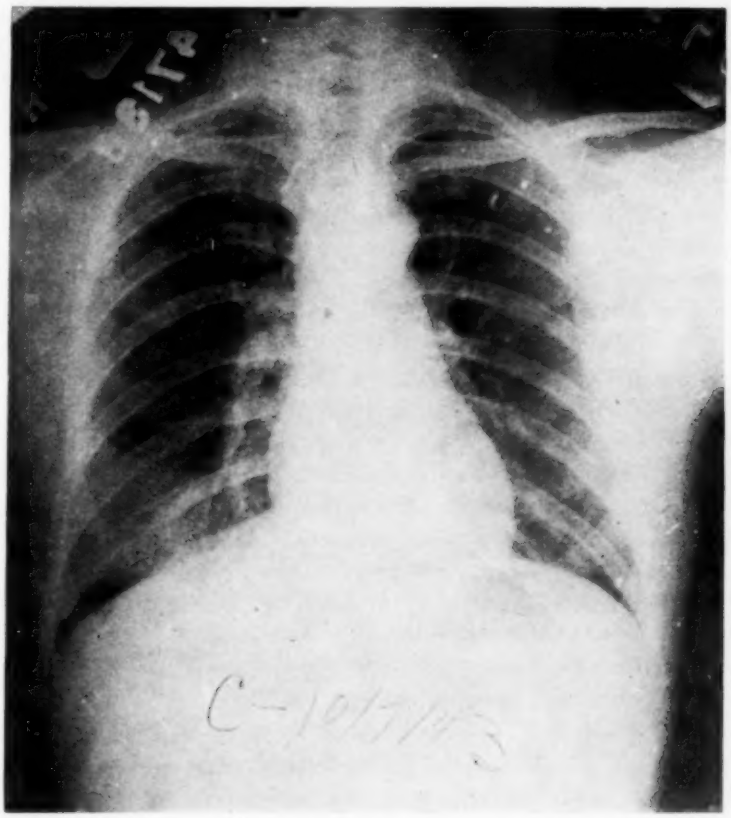

FIG. 5. Roentgenologic characteristics of the pulmonary involvement.

1 : 224. The second patient showed a small area of pneumonitis at the base of the left lower lobe, extending from the heart border to the periphery and his titer remained stationary at 1 : 112. The titer rose from 1 : 112 to 1 : 224 in the last patient, who had an involvement of the medial portions of both lower lung fields. Abnormal lymphocytes could not be found on repeated examinations, a mononucleosis never developed and the Davidsohn absorption test was negative in the two cases in which it was performed. It is evident, therefore, that differentiation between the two conditions would be im-

possible in many instances without the aid of the hematological picture. Although the pulmonary infiltrations cleared very rapidly, this phenomenon can occasionally occur in atypical pneumonia. During the course of this epidemic and especially during the winter months, sporadic cases of atypical pneumonia were admitted to the hospital. In none of these were atypical lymphocytes demonstrated.

The possibility of a secondary bacterial invasion must also be considered. Against this were the absence of significant organisms on sputum typing and culture, the clinical course, the roentgen-ray findings and the lack of the expected polymorphonuclear leukocytosis. In view of the systemic nature of the disease, there seems every reason to believe that a pneumonitis, closely resembling atypical pneumonia, can occasionally be due to the unknown etiologic agent of infectious mononucleosis.

7. Glands. Palpable glands were present in practically all our cases, including the insidious ones. It was, of course, impossible to state in many whether the glands were actually enlarged without knowing what they were normally like. There were, however, several aids in arriving at a decision. These were tenderness of the nodes, subsequent further enlargement or decrease in size, enlargement of glands in unusual regions and progressive involvement of different sites. When present, the glands were palpable on admission in the great majority. In a few cases, they did not appear until later in the course of the illness. Not infrequently, there was a sudden increase in the size of the glands either during the febrile period or during convalescence, in one case as late as 21 days after the onset of the illness. In others the enlargement was associated with a relapse. A progressive involvement of different sites was rarely observed, but when it did occur, it was quite striking. As hospitalization was for the most part of short duration, the glands were still palpably enlarged on discharge in the great majority. The glands usually began to shrink with the subsidence of fever, but in some patients who reëntered the hospital for various reasons, they were found to be enlarged as much as six months later.

The glands were usually small, about the size of a lima bean, but were occasionally larger. The largest gland in this series was in a soldier with a German measles syndrome who had an anterior cervical node near the angle of the jaw the size of a hen's egg. They were firm, elastic and discrete. Tenderness was usually slight but not invariably so. In 11 cases, the cause of admission was the tender, swollen glands. Six of these complained solely of the cervical glands, three of both the cervical and inguinal glands, one of the cervical and axillary glands and one of the inguinal glands alone.

A considerable number complained of pain in the back of the neck and at the base of the skull. In some of these cases the tender enlarged posterior cervical and occipital glands were associated with a defensive spasm of the neck muscles and a resulting stiffness of the neck. As headache was also a prominent symptom, these cases were difficult to differentiate from meningococcus meningitis or a meningitis complicating infectious mononucleosis.

There was usually no reaction in the overlying skin. A slight edema, however, was occasionally present. When this occurred over enlarged pre-auricular and mandibular glands in two of our cases, they were mistaken for mumps.

The anterior and posterior cervical nodes were the most frequently involved. We did not notice any predilection for the left side. In order of frequency, the sites of the glandular involvement were as follows: posterior cervical, anterior cervical, axillary, inguinal, femoral, occipital, epitrochlear, submental, supraclavicular, popliteal, pre-auricular, post-auricular. Multiple regions were involved in almost all who presented glandular enlargements of any extent. Although we suspected mediastinal glandular involvement in a number of cases because of the presence of bronchitic râles or because of a spasmodic cough, we were unable to demonstrate them by roentgen-ray in any of the 53 patients so examined. Neither have we been able to palpate enlarged mesenteric glands in those with abdominal pains or with icterus.

Suppuration, attributable to infectious mononucleosis, did not occur in a single case. In one patient, who had a suppurating inguinal lymph node on admission, biopsy revealed that the disease was complicating a preëxisting lymphogranuloma inguinale.

8. Gastrointestinal System: The frequency of anorexia has already been mentioned in the discussion of the initial symptoms. Nausea and vomiting were uncommon and were almost wholly confined to the icteric and abdominal varieties. Constipation was the rule. There were only 27 cases who had a diarrhea either at the onset of the illness or during its course.

An interesting group was the 12 cases whose major symptom was abdominal pain. In eight of these, the pain and tenderness were confined to the right lower quadrant or shifted to this region from the epigastrium. Nausea and vomiting were frequently present. All of these cases were admitted with the diagnosis of acute appendicitis. Fortunately, the blood count on admission in all of them showed the abnormal lymphocytes and none was operated upon. In two each, the abdominal pain was generalized or confined to the right upper quadrant. In one unusual case, not included in the abdominal variety, the patient was admitted with chills, fever, headache, malaise, cough and pains in the anterior chest of two days' duration. A moderate diffuse pharyngitis, generalized glandular enlargement and a barely palpable spleen were the only findings. On the third hospital day, he complained of severe abdominal pains beneath both costal margins and a mass was palpated in the right lower quadrant. In the next two days it had increased so markedly that it was visible on inspection of the abdomen and extended to the right costal margin. Operation revealed a huge mesenteric chylous cyst which had undergone degenerative changes.

9. Liver. The liver was palpable in 17 per cent or half as frequently as the spleen. Usually, it was described as extending a finger's-breadth below the costal arch and not tender. In the others, the enlargement was two to

four fingers'-breadth and tenderness was present on palpation. The latter findings were almost invariably present when jaundice was manifest.

Mackey and Wakefield [41] are usually credited with being the first to describe jaundice in infectious mononucleosis in 1926. Three years earlier, however, Downey and McKinlay [42] reported transient jaundice in one of their nine patients. Since then, there have been reports of isolated cases and of the occurrence of icterus in collections of sporadic and epidemic cases. In 1944, Spring [43] was able to compile 35 such cases from a partial survey of the literature, exclusive of five of his own patients. We have been able to find 25 additional cases.[42, 7, 44]

The actual frequency of this manifestation is difficult to compute for a number of reasons. In groups of reported cases, the incidence of jaundice has varied as follows: Bernstein,[10] 1.5 per cent; Press, Shlevin and Rosen,[44 (n)] 5.2 per cent; Stuart et al.,[28] 7 per cent; McKinlay,[45] 9 per cent; Spring,[43] 13 per cent; Nyfeldt,[46] 15 per cent. Collections of sporadic cases, however, have been reported in which jaundice did not appear.[32, 33, 47] In the recent epidemic described by Halcrow and his co-workers,[2] jaundice did not occur, although latent jaundice was present in eight of 15 severe cases. Besides these variations in incidence, many of the reported cases cannot be accepted as unequivocal. In view of the frequent occurrence of a marked lymphocytosis and a high percentage of atypical lymphocytes in infectious hepatitis,[8, 9] this condition cannot be excluded in most instances without a Paul-Bunnell test or a Davidsohn absorption test when the heterophile antibody agglutination titer is low or border-line. Increased sheep-cell agglutinins have not been reported in infectious hepatitis.[9] This objection applies not only to those cases reported prior to Paul and Bunnell's publication [12] but to many of the subsequent cases. In the latter, a heterophile antibody agglutination either was not performed or was negative or it was impossible to determine from the information given, whether the seropositive cases included all patients with jaundice. We do not wish to infer that there have been no authentic cases of jaundice in infectious mononucleosis. In fact, Martin [44 (c)] was apparently the first to describe a positive absorption test in one of his cases.

There were 34 cases (6 per cent of our series) with jaundice. The degree of icterus on admission varied considerably and depended both on the severity of the underlying process and on the length of time which had elapsed between the onset of symptoms and entrance into the hospital. Several cases were admitted at the stage in which the jaundice was subsiding.

DeVries [48] has classified the cases of infectious mononucleosis associated with jaundice into three groups: (1) Jaundice is the first symptom and is then followed by glandular enlargement. (2) Jaundice appears simultaneously with the glandular enlargement. (3) Jaundice with or without fever is the only symptom. Such a classification may be feasible in sporadic cases that are under close observation by a single observer from the onset of the disease. In this series, many cases were admitted late in the illness, while in

others, slight glandular enlargement was frequently overlooked until the blood picture called attention to the correct diagnosis.

Clinically, we were able to divide our cases into two distinct groups. In the first, consisting of 19 patients, there was no sore throat with the onset of the illness. In three of these a sore throat developed during the course of the disease; in one, as late as 20 days after admission. It is, of course, possible that a mild sore throat may have been present in some of these and forgotten by the patient. The usual history was that of anorexia, malaise, nausea and vomiting, followed in two to five days by jaundice. There was no fever or the temperature remained below 100° F. in 15. In addition, the three cases who developed a sore throat after the appearance of icterus were afebrile until this manifestation occurred. Among the symptoms that were occasionally present were pains in the right upper quadrant or epigastrium, diarrhea, chilly feelings and headache. In many of these no significant glandular enlargement was reported.

In the second group of 15 patients sore throat was always present at the onset. Fever, chills and headache were prominent complaints in addition to the symptoms described for the first group and the patients were usually hospitalized much earlier. The jaundice was also noticed two to five days after the onset. The glands were always palpably enlarged on admission.

It was our impression that the clinical picture for the entire group was indistinguishable from the ordinary case of infectious hepatitis with two minor exceptions. The icterus cleared somewhat more rapidly and the gastrointestinal symptoms were milder. The average duration of the jaundice in the 31 cases in which the onset of this manifestation could be determined with a fair degree of accuracy was only 25 days. However, the jaundice lasted for over 40 days in three, the longest period being 48 days. The shortest duration was 11 days. The well-being of the patients after the initial few days was notable. All were anxious to return to duty long before the jaundice had completely cleared. The vague digestive disturbances so frequently seen in the convalescence from catarrhal jaundice were conspicuously absent. There have been no demonstrable sequelae in those cases that we have been able to observe for as long as nine months.

As judged by the peak of the icteric index, three-fourths of the cases had jaundice of moderate severity. Excluding two cases admitted during the subsiding phase on the seventeenth and twenty-first days of their illness, the highest icteric indices recorded ranged between 24 and 100. Divided into groups, the results were as follows: 21 to 40, 6 cases; 41 to 60, 16 cases; 61 to 80, 8 cases; 81 to 100, 2 cases.

The heterophile antibody agglutination titer was 1: 112 or over in 25 cases. There were three cases with titers of 1: 1792. In the other nine there was either a rising titer to 1:56 or a positive absorption test.

As autopsy findings in infectious mononucleosis have been but rarely reported, an attempt was made to gather information on the nature of the liver involvement and its severity by a number of liver function tests.

The van den Bergh reaction was positive direct and indirect in all the 27 cases in which this test was performed.

The cephalin-cholesterol flocculation test as described by Hanger was done in 25 cases. The results were as follows: 0, 1 case; 1 plus, 1 case; 2 plus, 3 cases; 3 plus, 14 cases; 4 plus, 6 cases.

The total plasma cholesterol was determined in 27 cases. The highest values varied between 110 mg. per cent and 354 mg. per cent. If 250 mg. per cent is taken as the upper limit of normal, 18 were normal and nine were increased. As repeated determinations are of much more diagnostic importance than a single determination, these were performed repeatedly during the course of the illness in 12 cases. There was a correlation between the degree of icterus and the total cholesterol level in all. A rising icteric index was associated with an increase in the cholesterol and vice versa.

In the two cases in which serum phosphatase determinations were done, the values were 11.4 and 14 Bodansky units per 100 c.c., respectively.

Bromsulfalein tests were performed in 16 cases. In three the dose employed was 2 mg. per kilogram of body weight. None showed retention of the dye in 30 minutes. The dose was increased to 5 mg. per kilogram of body weight in the remaining 13 cases. In 10 of these there was a retention of the dye at the end of 60 minutes, varying between 20 per cent and 44 per cent. Only two of the 10 showed values below 30 per cent. The test was not performed until late in the course of the disease in the other three. Their icteric indices at the time of the test were 28, 21 and 11, and the retention of the dye in 60 minutes was 10 per cent, 10 per cent and 5 per cent respectively.

Glucose tolerance tests were given to 11 patients. In several of these the test was repeated at varying stages of the illness and in convalescence. The dose of glucose was 100 gm. and the criteria employed were a normal fasting blood sugar, a peak not exceeding 170 mg. per cent and a return to the initial level in two hours. Abnormal curves were found in nine, most of them markedly so. The degree of abnormality paralleled the course of the disease and returned to normal in convalescence. The two normal tests were obtained in patients in whom the jaundice was rapidly subsiding. All patients had been on a high carbohydrate, low fat diet for varying periods prior to the tests, which may have influenced their response.

In the 12 cases in which quantitative urinary urobilinogen determinations were performed, urobilinogen was present in all but not in abnormal amounts. The stools were never completely acholic, though fecal urobilinogen determinations were not done.

In summary, both the clinical picture and the liver function tests would indicate that the jaundice of infectious mononucleosis is due to a diffuse hepatitis, which is accompanied by an obstruction of the bile capillaries and a derangement of many of the functions of the liver. In the literature on jaundice in infectious mononucleosis, there are statements by several authors [41, 44 (h), 44 (n), 48] that the icterus is secondary to an obstruction by enlarged lymph nodes, although no evidence is given for such an assumption.

There is, however, evidence that a diffuse hepatitis does occur in infectious mononucleosis.

In a case recently reported by Ziegler,[44 (1)] in which death was due to a rupture of the spleen, the liver showed a diffuse focal hepatitis. Scattered throughout the liver were perilobular foci, composed of swollen Kupffer cells and numerous mononuclears. These were most prominent in the portal areas. There were destruction and disappearance of most of the liver cells in these foci. The small bile ducts were also involved. Many showed pericellular edema with separation of the duct epithelium from the basement membrane and others were lined with swollen epithelium which diminished or completely obliterated the lumen.

Even in the absence of jaundice the liver is frequently palpable. When the enlargement is marked and associated with a palpable spleen, the syndrome has been called the visceral form of infectious mononucleosis.[1 (d)] It would be difficult to explain such a phenomenon on any other basis than a diffuse hepatitis. In this connection, Van Beek and Haex[49] performed a liver biopsy in a case of infectious mononucleosis without jaundice, 14 days after the onset of the illness, and found the liver cells to be interspersed with monocytes and some polymorphonuclear leukocytes. The triangles of Kiernan were rich in cells and contained numerous lymphocytes. Another puncture, performed three and one-half weeks later, revealed the liver to be practically normal except for increased lymphocytes in the triangles of Kiernan.

10. Spleen. The spleen was palpable in 35 per cent of the cases. It was usually felt about a finger's-breadth below the costal margin on deep inspiration and never exceeded three fingers'-breadth. Because of the difficulty in palpating a spleen that was only slightly enlarged, the fact that the splenic enlargement was not always present on admission but appeared later in the illness and therefore had to be persistently sought for to be discovered, and because the cases were under the care of different officers of variable experience, this percentage is far from accurate. The enlargement had rarely disappeared by the time the patient was discharged from the hospital and, in some cases, it was still palpable three months after the initial illness. Rarely, the spleen was tender on palpation.

CASE REPORTS

ACQUIRED SYPHILIS OF THE LUNG: REPORT OF A CASE WITH AUTOPSY FINDINGS AND DEMONSTRATION OF SPIROCHETES *

By Joseph M. Wilson, M.D., *Los Angeles, California*

Acquired syphilis of the lung is an uncommon manifestation of syphilis according to many reports, particularly those written by the pathologist. Although well over 200 cases have appeared in the literature, only a minority have been confirmed by autopsy. A considerable difference of opinion concerning the diagnosis and incidence of pulmonary syphilis exists not only between the clinician and the pathologist, but also among the pathologists. This controversy can doubtless be attributed to the relatively non-specific clinical features, to the variation in diagnostic criteria employed, and lastly to the fact that such a lesion may easily be overlooked at the autopsy table because of its obscure nature and the presence of superimposed secondary infection.

Funk [7] (1923) found only four cases of pulmonary syphilis among 1,200 patients admitted to the tuberculosis wards of the Jefferson Hospital; on the contrary, Kirkwood [14] (1926) believed that about 3 per cent of all cases admitted to tuberculosis sanatoria had an associated syphilis of the lung. Claytor [3] (1905) reported not a single case of pulmonary syphilis in 13,000 specimens at the Army Museum at Washington, D. C. Symmers [25] (1916) found 12 cases of lung syphilis in 4,800 autopsies, whereas Carrera [2] collected 12 cases in the study of 152 autopsied cases of syphilis. Of 6,748 autopsies performed in a recent three-year period at the Los Angeles County Hospital, I could find but two cases of acquired pulmonary syphilis, although 132 cases showed other anatomical lesions of syphilis. Howard [11] encountered seven cases of pulmonary syphilis in a total of 11,982 medical admissions to his clinic; four of these cases were confirmed by autopsy. Among 3,427 syphilitics at King's County Hospital, Denman [5] reports five cases of pulmonary syphilis, four of which were proved by necropsy. In China, Lieu [17] has found two cases of pulmonary syphilis in 2,800 autopsies. It is thus obvious that both clinicians and pathologists are far from being in agreement as to the true incidence of acquired pulmonary syphilis.

The following case meets the accepted criteria for the diagnosis of pulmonary syphilis, including the demonstration of the morphologically typical *Treponema pallidum*.

Case Report

E. E., a 69 year old Mexican male newspaper vendor, entered the Los Angeles County Hospital on Aug. 15, 1944, complaining of generalized progressive weakness for one year, mild weight loss and frequent cough for several months. The cough

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was productive of foul, whitish-yellow mucoid sputum averaging one cup daily. For several weeks prior to admission, the patient had exertional dyspnea and an intermittent pain between the shoulder blades which was aggravated by coughing. There had been no hemoptysis, fever, or chills, and only occasional night sweats. Patient had been able to work until two days before admission. There were no tuberculous contacts, and venereal disease was denied by name and symptom. The past history was not contributory.

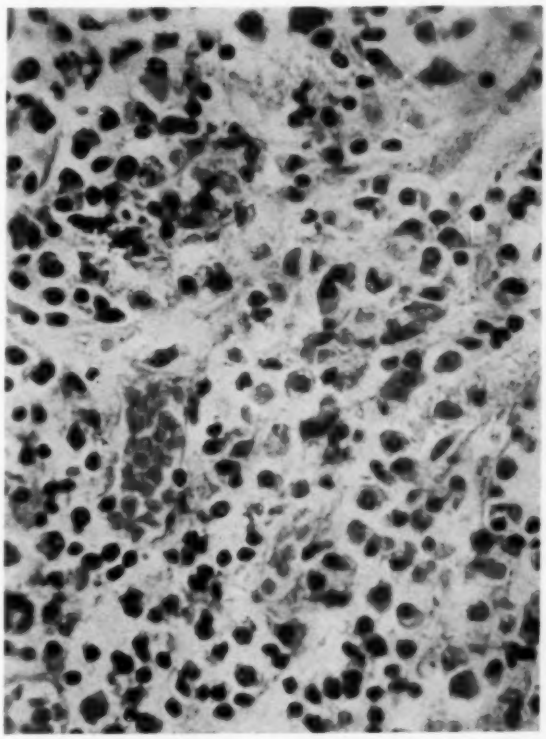

FIG. 1. Lung. Infiltration of plasma cells and lymphocytes with mild fibrosis in an active syphilitic lesion. H. & E. stain. × 230.

Physical Examination. The patient was well developed, well nourished, and appeared mildly dyspneic and orthopneic. The temperature and pulse were normal. The respirations were normal in rate. The skin was clear. Examination of the eyes, ears, nose, and throat was negative. The trachea appeared in the midline. Prominence of the right side of the chest was noted, both anteriorly and posteriorly. The veins in the suprasternal notch were distended. The right lung field was dull to percussion over the lower one-half posteriorly, and increased vocal and tactile fremitus were evident together with numerous crackling râles and increased breath sounds in the same region. The left lung field was normal. The heart was enlarged, and the apex beat was palpated 2 cm. to the left of the midclavicular line. Normal sinus rhythm was present and no murmurs were heard. The blood pressure was 120 mm. Hg systolic and 60 mm. diastolic. The abdomen was negative. The fingers and toes showed a moderate degree of pulmonary osteoarthropathy. The deep reflexes were hyperactive, and there were no pathological reflexes.

Laboratory. Examination of the blood revealed a red cell count of 4,620,000 with hemoglobin of 83 per cent. The white-cell count was 11,000 with neutrophiles 68 per cent, lymphocytes 32 per cent, monocytes 4 per cent, and eosinophiles 2 per cent. The corrected sedimentation rate was 19, by the Wintrobe method. The blood Wassermann and Kahn reactions were four plus on two occasions. The urine was normal. The sputum was negative for tubercle bacilli on at least three examinations. The tuberculin skin test was positive with No. 3 O.T. (0.1 mg.) The coccidioidin skin test was negative (1–100 dilution).

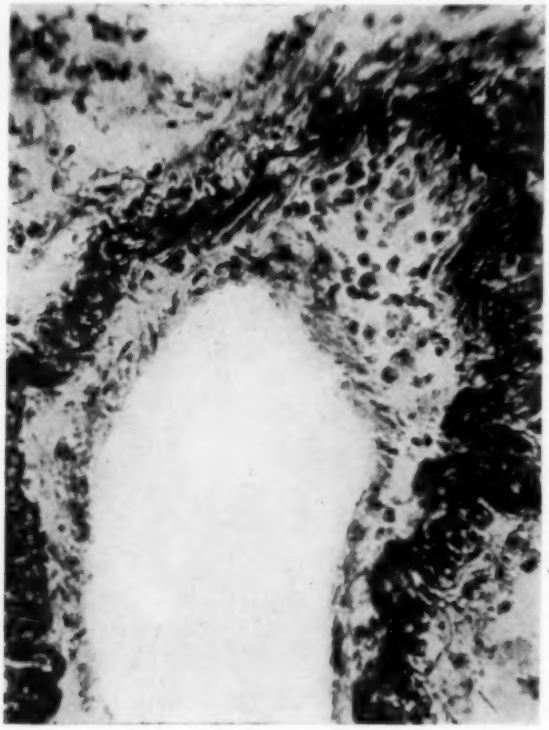

FIG. 2. Pulmonary vessel with characteristic cellular infiltration of intima and adventitia, and widening and splitting of the internal elastic membrane. Krajian's elastic fiber stain. × 120.

Course. The patient had a low grade fever, rarely above 99° F., but occasionally rising to 100° F. The initial roentgenogram of the chest taken Aug. 17, 1944, revealed a semi-confluent infiltration in the right perihilar region with marked thickening of the right hilum and diffuse involvement in the right lower lobe (figure 5). The patient continued to have a productive cough and showed little improvement. Bronchoscopy was done Sept. 8; the carina appeared markedly broadened and no evidence of intra-tracheobronchial tumor was found. A moderate amount of mucopurulent material was seen in the right main bronchus, originating chiefly from the lower lobe orifice. Lipiodol study of the right lung showed a fairly normal bronchogram in the upper and lower lobes, but very little lipiodol entered the right middle lobe. Pulmonary syphilis was considered in the differential diagnosis, and the patient was placed on potassium iodide, minims 15 three times daily. Ten days later, the roentgenogram showed no reduction in the right mid-lung density. On Oct. 28, the patient had im-

proved sufficiently to be discharged to his home, and was instructed to continue potassium iodide therapy. The patient visited the clinic on Nov. 30, and complained of a constant and severe cough which no longer responded to potassium iodide therapy. He had had a single episode of hemoptysis three weeks previously, but none since. A roentgenogram (figure 6) taken in the clinic revealed a slight decrease in density of the infiltrated area in the right mid-lung field as compared with the previous film of Oct. 19. The patient was advised to continue potassium iodide and mild sedation. On Dec. 5, the patient was readmitted to the hospital because of frequent episodes of hemoptysis, increasing dyspnea and orthopnea, and progressive loss of weight. On

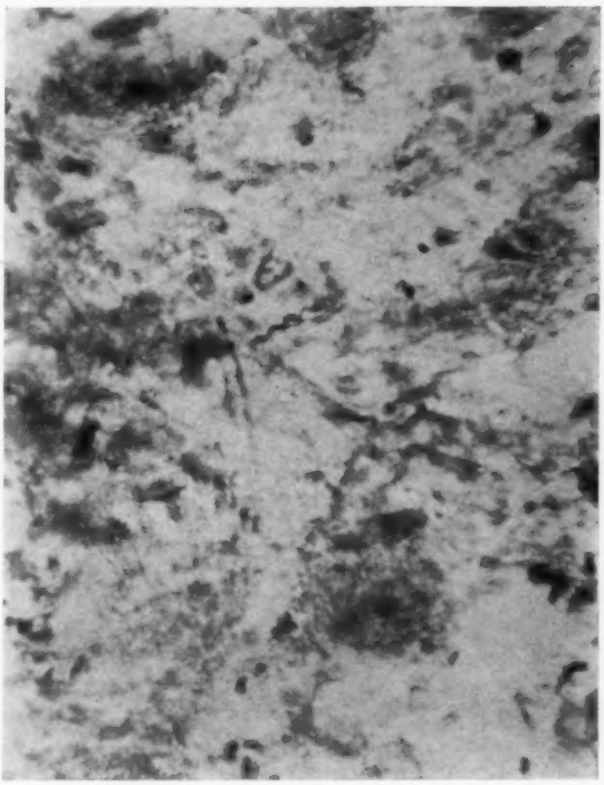

FIG. 3. *Treponema pallidum* in section of lung tissue. Several others were present in this field at different levels of focus. Krajian's spirochete stain. × 1770.

admission, the temperature was 102° F., and the patient was very dyspneic and orthopneic. The middle and lower portions of the right lung field were dull to percussion anteriorly, laterally, and posteriorly. Harsh breath sounds which were almost amphoric in quality were present over the right upper lobe posteriorly. Numerous crepitant râles were heard throughout both lung fields. Other physical findings were unchanged from the previous admission. The cough remained constant, with production of greenish-yellow sputum which was again negative for the tubercle bacillus. The temperature returned to normal for a few days after admission, but thereafter rose frequently to 99° and 100° F. No further roentgen study was made. On Jan. 26, 1945, the patient developed a temperature of 104° F. with signs and symptoms

of bronchopneumonia. Sulfadiazine and supportive therapy were given without response, and death occurred on Jan. 31, 1945.

Postmortem Examination. Necropsy was performed by the author five and one-half hours after death. Externally there was evidence of marked weight loss and a moderate degree of pulmonary osteoarthropathy of the extremities.

The heart was normal in all respects except for the aortic valve which showed slight widening of the commissures. The coronary ostia were of normal size. The aorta contained numerous white and yellow raised patches of tree-barking, together with longitudinal wrinkling of the intima. A moderate degree of atherosclerosis was

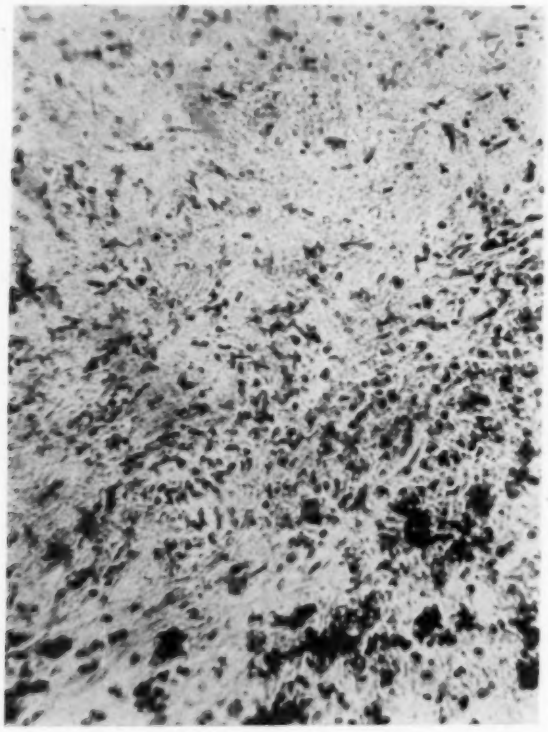

FIG. 4. Bronchial lymph node containing a portion of a small gumma. The central caseous zone contains nuclei in varying stages of karyorrhexis, and is bordered by an infiltration of lymphocytes and plasma cells. H. & E. stain. × 120.

present, particularly in the abdominal portion of the aorta. Microscopic examination of the aorta revealed an infiltration of plasma cells and lymphocytes around the vasa vasorum of the adventitia and outer portion of the media. The media contained areas of fibrosis.

Lungs: The right lung weighed 1050 grams, and the left lung weighed 900 grams. Numerous fibrous pleural adhesions were present over both upper and middle lobes of the right lung, and fibrinous adhesions were found over the right and left lower lobes. Both lungs were subcrepitant to palpation. The perihilar region of the right lung was quite firm to palpation. A single enlarged lymph node measuring 2 by 2 cm. was found near the origin of the right middle lobe bronchus. On section this node showed an excessive amount of black pigmentation in addition to several small, grayish-white, and relatively firm areas. On section of the right lung, the perihilar regions of the

middle lobe and lower portion of the upper lobe contained several large, firm, rubbery masses. These masses were fairly well circumscribed, exhibited a fine mesh-like structure, and appeared diffusely grayish-pink in color except for scattered anthracotic deposits. The larger perihilar mass located in the middle lobe, measured 3.5 by 5 by 3.0 centimeters; it was intimately attached to the adjacent bronchi and blood vessels, and extended out along these structures toward the pleura. Dense fibrous bands radiated in all directions from this central mass (figure 7). Just beneath the pleura in the upper portion of the right lower lobe was found a small, thick-walled

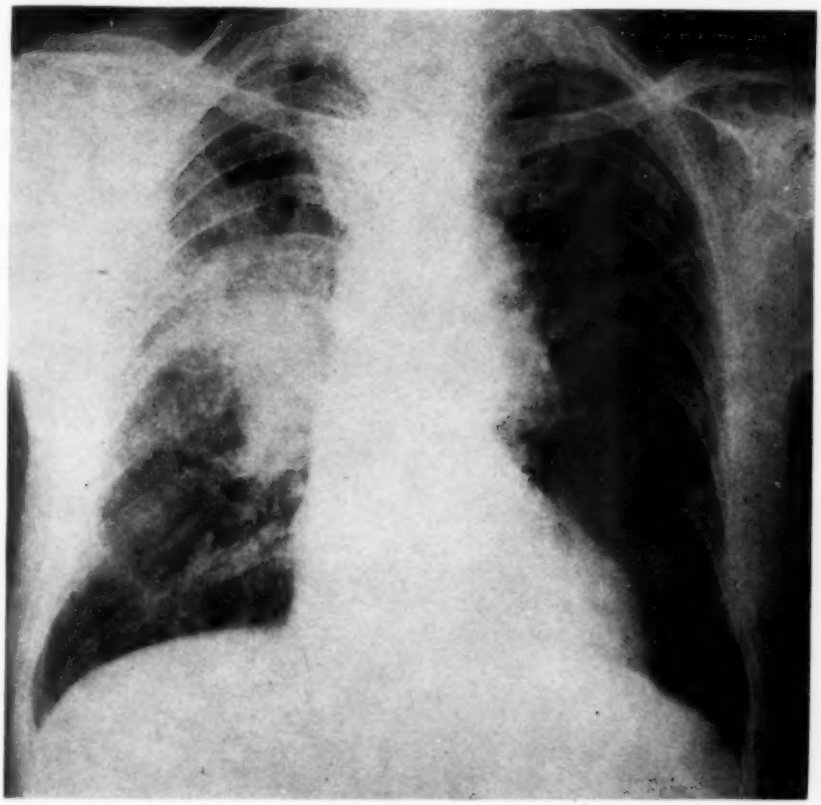

FIG. 5. Initial roentgenogram. Note the dense infiltration of the right midlung field, with strand-like projections radiating toward the pleura.

bronchiectatic cavity containing a small amount of mucopurulent material. Throughout the left and right lower lobes there was gross evidence of bronchopneumonia.

Microscopic Examination of Lungs: Sections of the perihilar mass in the right lung revealed areas of fibrosis varying in density, with complete obliteration of the alveolar structure and infiltration of myriads of plasma cells and lymphocytes (figure 1). Anthracosis was marked in this area, and relatively few capillaries were present. No areas of frank necrosis were found in the parenchyma. In the bronchioles there was a mild degree of mural thickening and dilatation, with purulent exudate in the lumina. There was infiltration of the intima and adventitia of the arteries by plasma cells and small lymphocytes. The abnormally prominent internal elastic membrane was well demonstrated by special elastic tissue stains (figure 2). The elastic tissue

stain also revealed the characteristic persistence of the elastic fibers throughout the involved parenchyma. The morphologically typical spirochetes were demonstrated by the Krajian [15] spirochete stain (figure 3). The spirochetes were found in moderate numbers in those areas of fibrosis showing the greatest activity. Sections of lung taken elsewhere revealed many areas of bronchopneumonia. The bronchial lymph node contained a number of small areas of gummatous necrosis with the central zone of necrosis surrounded by an exceedingly vascular fibrous connective tissue layer and many small lymphocytes and plasma cells (figure 4). Acid-fast stains of this section

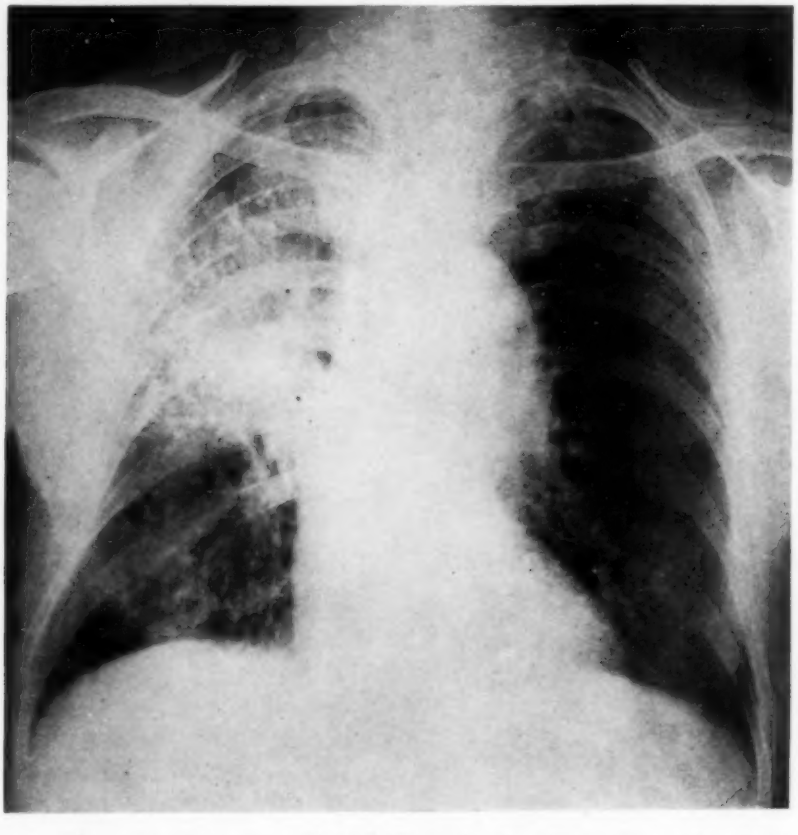

FIG. 6. Later roentgenogram. Right midlung density after seven weeks of potassium iodide therapy. Minimal regression has occurred since initial film. Some lipiodol remains in the right upper lobe from previous lipiodol study.

of the lymph node were negative for the tubercle bacillus. The postmortem examination revealed no other positive findings.

Anatomical Diagnosis: Syphilis of right lung, acquired; gummata of bronchial lymph node; bronchiectasis; syphilitic aortitis; bronchopneumonia.

CLINICAL FEATURES

The clinical symptoms of pulmonary syphilis are those of any chronic pulmonary infection, and most often simulate and suggest pulmonary tuberculosis.

The symptoms in order of apparent frequency are: (1) Cough, usually of a constant and troublesome nature, with production of mucopurulent sputum in varying amount which is repeatedly negative for tubercle bacilli. (2) Dyspnea is a common symptom, often out of proportion to the physical signs, and may be due to affection of the vagus nerve. (3) Hemoptysis is relatively uncommon as compared to its incidence in tuberculosis, but may be one of the outstanding symptoms as it proved to be in the author's case. Greenfield (quoted by Howard) has said: "From evidence afforded by other cases, I am inclined to believe that a special tendency to profuse hemoptysis with slight lung disease in cases of syphilis, is dependent partly on the high vascularity of the connective tissue growth in its early stage and partly on constriction of veins by surrounding thickening." The absence of fever, profuse night sweats, and severe loss of weight is characteristic of the pulmonary lesion of syphilis, unlike that of tuberculosis. Chest pain occurs infrequently, and is produced by the involvement of the mediastinum or pleura.

The physical signs are not distinctive, and only serve to confirm the presence of a pulmonary lesion. In some cases the physical signs are minimal, but in most cases the physical examination and roentgen study reveal evidence of consolidation and infiltration of the lung fields far out of proportion to the relative well-being of the patient. This discrepancy tends to favor the diagnosis of syphilis rather than tuberculosis. The right middle and lower lobes and the left lower lobe have been designated as the most common sites of localization by many writers, although Karshner and Karshner (quoted by Howard) found upper lobe involvement to be about twice as common as lesions in the bases and right middle lobe.

The roentgenological evidence of pulmonary syphilis is at best inconclusive, although it is a necessary and valuable adjunct to the clinical diagnosis. The presence in a syphilitic patient of a persistent unilateral hilar or lower lobe density with fibrous strands extending out toward the pleura, deserves serious consideration as a manifestation of pulmonary syphilis; however, other more common disease processes produce a similar picture, namely pulmonary tuberculosis, mediastinal neoplasms, bronchiectasis, pneumoconiosis, mycotic infection, and unresolved pneumonia. Warring [26] declares that roentgen-ray is an "inadequate differentiator" of pulmonary syphilis and is convinced that this condition cannot be diagnosed clinically. Serial roentgenograms are essential in determining the response of the pulmonary lesion to antisyphilitic therapy.

Pathology

Almost all of the pulmonary lesions of syphilis observed at autopsy are of the late, or tertiary, stage. Secondary syphilis may involve the mucous membrane of the bronchi, as illustrated in a case with bronchoscopic examination reported by Ornstein.[20]

Most authors agree upon the division of the lesions of acquired pulmonary syphilis into two general groups: (1) gummata, and (2) diffuse interstitial fibrosis. In his comprehensive treatise on pulmonary syphilis, Howard describes five forms as follows: (1) Gummata—the most readily recognized form, but less commonly found than formerly supposed. It is characterized by a central caseous area surrounded by a layer of vascularized fibrous tissue with an outer

layer of plasma cell and lymphocytic infiltration. (2) Chronic interstitial pneumonia, or peribronchial fibrosis, is the more common type and is similar to any chronic non-specific pneumonia. (3) Pulmonary sclerosis, or fibroid induration. This probably represents a more advanced stage of chronic interstitial pneumonia, with hard fibrous bands and nodules arranged along the bronchi, vessels, and interalveolar septa. The case herein presented best fits into this category. (4) Syphilitic phthisis is a mixed form of pneumonia, gummata, fibrosis, and cavitation.

The early stages of pulmonary syphilis have rarely been observed. Stanley (according to Carrera) has stated that in the first stages there is a diffuse mediastinitis, with intense cellular infiltration filling the alveoli, the septa, the peribronchial and perivascular tissue, with epithelial desquamation, giving a gelatinous appearance to the portion of the lung involved. Letulle, in a publication by Courcoux and Lereboullet,[4] spoke of the "follicule elementaire" as being characteristic of the early stage of syphilis; this formation is described as a collection of spirochetes, lymphocytes, and plasma cells located in the interstitial tissue around the small bronchioles, blood vessels, and in the interalveolar septa. Letulle considers this formation to be the initial phase in the evolution of the gumma.

Many authorities have considered the gumma to be the most characteristic and important lesion of late syphilis. However, in Warthin's[27] opinion, the gumma is neither the most typical nor the most common syphilitic lesion in the lung or elsewhere. His concept of the pathology of syphilis is based upon the demonstration that the essential lesion of late syphilis results from the instigation of a specific and progressive inflammatory process, usually mild in degree, characterized by lymphocytic and plasma cell infiltration in the stroma. This infiltration occurs particularly about the blood vessels and lymphatics, with subsequent atrophy or degeneration of the parenchyma. The elastic fibers throughout the involved lung tissue are not destroyed, and can be demonstrated in great numbers by special elastic tissue stains. Scarring and thickening of the pleura may occur, and pleural effusion has been reported, such as the case described by Hu, Frazier, and Hsieh.[12]

In pulmonary syphilis there are characteristic changes in the pulmonary vessels. There commonly occur an endarteritis and periarteritis without appreciable damage to the media. Plasma cells and lymphocytes infiltrate the intima and adventitia; the internal elastic membrane is rarely destroyed, and usually becomes abnormally prominent with widening and splitting of this membrane. This is in contrast to the changes produced by tuberculosis, consisting of damage to all three coats of the vessel, including the destruction of the elastic fibers.

Carrera has maintained that it is only in the presence of the active, specific inflammatory process, due to the local action of the spirochete, that the positive diagnosis of syphilis of the lung can be made. If the local syphilitic process has become completely inactive, this residual fibrosis cannot be differentiated from a host of other types of pulmonary fibrosis. As long as the active lesion persists, there is reasonable assurance that the *Treponema pallidum* is present in some form. In the past only a few investigators have reported the identification of the spirochetes in lung tissue, and in many cases no attempt has been made to find them. Those reporting the presence of the spirochete include Henske (1918),[10]

Kielty (1916),[13] and Warthin (1917).[28] McIntyre [18] stated that both Koch (1907) and Vogelsang (1929) demonstrated the organism, and Howard credits Schmorl and Levaditi with this accomplishment. Windholz (1929) [29] searched in vain for the spirochete, but noted the presence of granular bodies within the giant cells which he thought might represent the changed or broken down form of the spirochete. It is important when looking for the *Treponema pallidum* to be mindful of the "ever-present pitfall of artefact," as Stokes [23] has aptly phrased it, and to ascertain that the organism is morphologically typical.

In view of the marked similarity of the anatomic changes in the lung produced by tuberculosis and syphilis, the differential diagnosis of these two lesions is most important. McIntyre has given an excellent summary of the important points in the differential diagnosis: (1) The caseating power of syphilis is more limited than that of tuberculosis. (2) Healed tuberculosis is circumscribed, whereas the lesions of syphilis show prolongations. (3) Giant cells are rarer in syphilis than in tuberculosis. (4) Plasma cells are rarer in tuberculosis than syphilis. (5) Anthracosis is more infrequent in the scar of syphilis than in the scar of tuberculosis. Carrera believed the opposite to be true. (6) The nodule of tuberculosis may arise without a "follicule elementaire," and may begin as an island of encapsulated bronchopneumonia. (7) The invasion of a scar with subsequent breaking down is less common in syphilis than in tuberculosis. (8) Blood vessels are more rarely attacked in syphilis. (9) The adventitia of the blood vessels is the portion that is more frequently affected in syphilis. (10) There is no epithelioid formation in syphilis as in tuberculosis. (11) The elastic tissue is better preserved in syphilitic than in tuberculous processes. (12) Syphilis produces a greater amount of connective tissue in the pleura than tuberculosis. (13) Tuberculosis does not show as many cortical pulmonary lesions adjacent to the affected pleura as does syphilis. Carrera studied the lungs of 60 cases of tuberculosis, noting the characteristic connective tissue formation and comparing it with that produced by syphilis; from this study he was convinced that it is never impossible to distinguish between these two types of fibrosis.

Diagnosis

Many cases of pulmonary syphilis appearing in the literature have been diagnosed on the basis of clinical evidence alone. The relative insecurity of attempting to confirm the diagnosis of this condition clinically must be borne in mind, although one is certainly justified in making a presumptive diagnosis based upon adequate clinical evidence. Hartung and Freedman [9] gave the following criteria for diagnosis of pulmonary syphilis: (1) History of syphilis, including evidence of stigmata of syphilis elsewhere in the body. (2) Signs and symptoms of a chronic, stubborn, and progressive pulmonary lesion. The signs are usually marked and the symptoms mild. (3) Repeated sputum examinations must be made to rule out tuberculosis, mycotic infection, and other spirochetal infections. (4) Demonstration of the *Treponema pallidum;* this is the ultimate proof. (5) Serologic examination. (6) Roentgenogram. The involvement of the root, middle lobe or bases of the lung is most suggestive. (7) The therapeutic test. (8) Anatomic confirmation whenever it is possible.

It has been stated by Allison [1] that even a presumptive diagnosis of pulmonary syphilis is not warranted until all other types of pulmonary disease have

been excluded. On the other hand, Flockmann (quoted by Denman) had previously maintained that pulmonary syphilis was the most probable diagnosis in a syphilitic patient with evidence of a chronically progressive pulmonary infiltration. This contention was upheld by Denman. As a general rule, it is wise to search thoroughly for the presence of the tubercle bacillus, for although tuberculosis and syphilis may be co-existent, it is most encouraging to know whether or not tuberculosis is confusing or complicating the picture.

The therapeutic test as a diagnostic aid has been relied upon quite heavily by some observers. Although this test admittedly offers helpful contributory evidence, I believe as many others do, that it should not receive too much emphasis; it is common knowledge that antisyphilitic therapy may cause regression and improvement in many non-syphilitic pulmonary lesions which have the same configuration on the roentgen film as does syphilis. Osler [21] declared that the therapeutic test was by no means conclusive. Stokes advises the use of only mercury or bismuth in performing the therapeutic test, since arsphenamine and iodides have too large a margin of non-specificity. Freedman and Higley [6] followed the clinical course of a syphilitic patient with suspected pulmonary involvement, and failed to note any response of the pulmonary lesion to antisyphilitic therapy. At autopsy a gumma, which is ordinarily amenable to therapy, was found to be the same size as shown on the initial roentgen film taken prior to treatment. In this particular case the gumma was surrounded by such a dense fibrous capsule that it was almost impossible for this lesion to have become reduced in size on serial roentgenograms. In such cases the therapeutic test would obviously be misleading.

Other antemortem diagnostic procedures have been suggested. Examination of the sputum for *Treponema pallidum* has been unsuccessful according to most authors. Munson [19] has mentioned the procedure of direct lung puncture at the site of the lesion, thereby obtaining material in which the spirochete may be demonstrated. He further states that this procedure is contraindicated when one is not certain of the presence of the tubercle bacillus or other virulent organisms. Hu, Frazier, and Hsieh advised the study of aspirated pleural fluid by darkfield examination and also by inoculation of animals. They were fully aware of the scarcity of organisms in the pleural fluid, but nevertheless thought that this procedure afforded the possibility of recovering the spirochete during life.

At autopsy, the diagnosis can be confirmed with the discovery of the fairly characteristic histological changes in the lung and the identification of the spirochete. In addition to staining sections of the pulmonary lesion specifically for spirochetes, the inoculation of rabbit testes or the chimpanzee with material from the lesion has been helpful in demonstrating the presence of the *Treponema pallidum*. Unfortunately the latter procedure was not attempted in the author's case.

Treatment

The treatment of pulmonary syphilis is in general that of any tertiary syphilitic lesion, namely by mercury or bismuth and iodides followed by arsenicals if the clinical status of the patient permits. Penicillin should be tried in view of recent reports by Stokes et al.,[24] and Goldman,[8] pertaining to the action of penicillin in late syphilis. One may question the advisability of administering antisyphilitic therapy in a suspected case of pulmonary syphilis, when a known active or

quiescent tuberculous infection is present. Both Skavlem [22] and Lecaplain [16] have stated that there is no reason to withhold antisyphilitic therapy in such a case for fear of adversely affecting the tuberculous process.

A successful response of gummatous lesions to specific therapy is usually obtained, and the prognosis is good unless complicated by a superimposed pneumonia, gangrene, or abscess. Howard stated that little or nothing can be expected from treatment in the sclerotic forms of pulmonary syphilis.

Summary

A proved case of acquired syphilis of the lung with demonstration of the spirochete has been presented together with a review of the literature pertaining to the incidence, clinical aspects, pathology, diagnosis, and treatment of pulmonary syphilis.

Acquired pulmonary syphilis doubtless occurs far more often than commonly supposed. No doubt the reported incidence of pulmonary syphilis would rise if this diagnosis would be considered more frequently in the presence of chronic progressive pulmonary infection associated with a positive serologic reaction and other stigmata of syphilis. Moreover, the pathologist could also boost the incidence by investigating thoroughly any atypical pulmonary fibrosis or scarring encountered in cases showing collateral evidence of syphilis.

When histological changes characteristic or suggestive of syphilis are found in the lung, a thorough search for the *Treponema pallidum* should certainly be made. The demonstration of the organism by appropriate staining methods and, if possible, by animal inoculation, aids in establishing an unequivocal diagnosis.

I am grateful to Dr. E. M. Hall and Dr. E. M. Butt for helpful suggestions and comment. I wish to thank Mr. A. J. Lonberg for making the microphotographs.

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MYXEDEMA—CONTROLLED BY THYROID EXTRACT FOR FIFTY-TWO YEARS: REPORT OF A CASE*

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WHEN the diagnosis of myxedema has been made and thyroid therapy has produced the usual spectacular return of the patient to normal life, the question as to ultimate prognosis is asked. The usual answer is a statement that as long as the patient continues to receive thyroid in adequate dosage she will continue to live out the rest of her life unhampered by the deficiency in the activity of her own thyroid gland and that she may plan and carry on her life as may any normal person. That this is true is borne out by the facts here reported which concern a woman who developed severe myxedema at the age of 35, was put on thyroid at the age of 39 and died when almost 92 years old.

This report is of interest because it describes what is probably the longest period of successful treatment of myxedema on record. The patient herself believed that she was the first person to receive this treatment in America. She was put on thyroid by Dr. Calvin S. May of New York in 1892, one year after the first patient was given thyroid treatment in England by the late Dr. Murray. (Dr. Murray's first patient was said to have died in 1919.) As will be seen in the case report given below she was, for her age, vigorous and determined to the

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last. At times it was necessary to reduce the dosage of thyroid in order to relieve the attacks of angina pectoris which, in the last decade of her life, became very troublesome to her.

A discussion of this patient's story and her condition when she had reached the age of 84 years is given by Dr. James Howard Means.[1]

Case Report

Mrs. E. C. B. was first examined by the writer on February 6, 1924. She was then 71 years old, a housewife, living quietly with her husband, a retired manufacturer. When seen she was suffering from a subacute upper respiratory inflammatory process. In this attack no abnormality of her heart or lungs was made out but her blood pressure was found to be 220 mm. Hg systolic and 100 mm. diastolic.

Her family history and past history were irrelevant. She was married in 1876 and a year later gave birth to a daughter, her only child. She remained normal up to 1888 when her child died of diphtheria. This was undoubtedly a severe emotional shock to the patient. During the next few months it was noticed that her face became swollen, her hair began to fall out and her legs became "enlarged and shapeless." She was said to be anemic and it was said that for two years she ate one pound of steak a day in an attempt to correct this condition. Finally her local physicians made a diagnosis of nephritis and her husband was informed that she could not live more than six months. She was then taken to New York where she was examined by the late Dr. Calvin S. May who made the diagnosis of myxedema. According to the patient's account, authenticated by other members of her family, her condition, both physical and mental, was at this time extreme and evidently represented a very advanced stage of hypothyroidism. Dr. May placed the patient on thyroid gland but the exact form in which it was given was not remembered by the patient or the family. The patient was under the impression that she was the first person to receive thyroid treatment in America and that her case was reported by Dr. May at some medical gathering, but a search of the literature for this report has been unavailing. A letter from Dr. May's daughter to the writer states that all the doctor's papers had been examined and destroyed and no reference to the patient, who was well known to her, had been found.

After being restored to what appeared to be normal health the patient continued on thyroid and lived without acute illness except occasional "bronchial colds" until seen by the writer in 1924. She stated that at one time she took three 5 grain tablets of thyroid (Burroughs & Wellcome) a day but that the dose had been reduced to one 5 grain tablet daily and that this dose had been continued for 30 years. Following the first examination in 1924 which was made at her home she was seen casually from time to time for minor conditions. On January 29, 1926, the following examination was recorded:

Complaint: Main complaint is fatigability. Dr. Leech refers patient because of headaches and question of general condition. *Physical Examination:* Height 65 in. Temperature 95.8. Blood pressure 188 mm. Hg systolic and 92 mm. diastolic. Weight 166 lbs. Pulse 62. Patient is a well developed and nourished lady of healthy appearance for her age. Skin is clear. Mucous membranes of good color. Eyes: Pupils equal, regular and react normally; fundi normal. Hair thick and gray; not dry; in good condition; no dryness of scalp. Thyroid is not palpable and not visible. No peripheral lymph node enlargement noted. Chest symmetrical and well developed. Sides move equally with respiration. No abnormal retraction. Heart: Percussion unreliable. Rate slow: 64. Rhythm regular; no murmurs. A_2 somewhat accentuated. Lungs normal throughout. Tongue clean. Teeth: All lower molars missing and only three upper molars present. Throat clear; Tonsils small. Musculature rather flabby. Abdomen is somewhat obese, tympanitic. No spasm or tenderness.

No organs palpable. Considerable subcutaneous fat. Deep reflexes normal. Extremities negative.

Diagnosis: Myxedema (controlled by thyroid); arteriosclerosis, general; hypertensive vascular disease.

During the 20 years in which she was under observation the following information was obtained: The heart rate was always slow, usually 54 to 60 per minute, and on many occasions it was found to be very irregular. This was supposed to be due to auricular fibrillation but the electrocardiograms, of which two are reproduced, show that a marked sinus arrhythmia was present. The blood pressure was always elevated, the lowest noted being 170 mm. Hg systolic and 70 mm. diastolic, and the highest 300 mm. systolic and 160 mm. diastolic during an attack of angina pectoris on April 1, 1935. On only two or three occasions was the systolic pressure found below

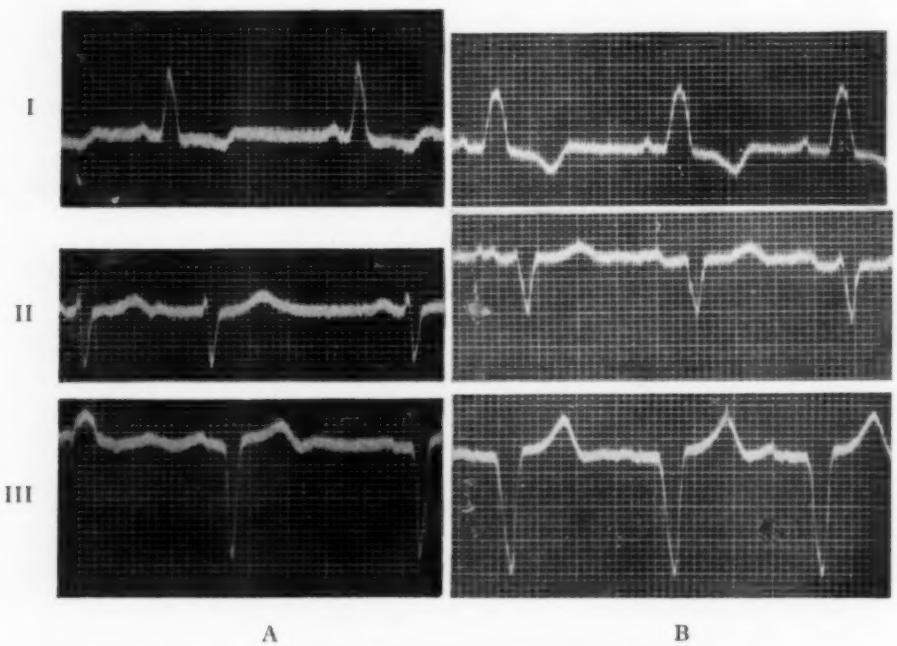

FIG. 1. A. Electrocardiogram taken in 1937; B. taken in 1940.

200 mm. Dyspnea on exertion was first noted in 1928, and basal pulmonary râles were found to be present at times in 1938. She had her first attack of severe precordial pain in February 1934, and from that time on she had periods in which such attacks were very annoying. They were usually promptly relieved by nitroglycerin. It was found in 1935 that by omitting her thyroid, or reducing its dose, the anginal attacks could be readily abolished but this was always followed by such depression and weakness that the patient invariably insisted on resuming her usual dosage. Eventually a dose was found (gr. 3½, Burroughs & Wellcome) which was low enough to keep her fairly free from angina and high enough to prevent her from developing the symptoms of myxedema which to her were equally to be dreaded.

The diagnosis of arteriosclerotic heart disease, evidenced by the anginal attacks, was also borne out by electrocardiographic studies which showed, besides marked left axis deviation, progressive interference with intraventricular conduction (figure 1).

There was no evidence of retinal damage or of renal failure. The blood non-protein nitrogen was found to be 32.7 mg. per 100 c.c. in 1935 and the blood sugar, at this same time, 101 mg. Blood cholesterol was found to be 222 mg. in 1941, blood Wassermann and Hinton reactions were negative and the blood sugar and non-protein nitrogen values were approximately the same as they were in 1935. Several urinalyses showed traces of albumin and clumped leukocytes in the sediment, and the patient at one time suffered a mild attack of dysuria. The specific gravity of the urine specimens examined varied between 1.007 and 1.020 and at no time were casts observed.

This patient was not easily managed, being quite dictatorial and constantly given to dismissing her nurses. She could only once be persuaded to have an estimation of her basal metabolic rate which was found to be minus 15 per cent. Requests were made that she be photographed for the record but she would never allow it. She remained in control of her household up to the last few months of her life. In 1939 she was able to go away for the summer but in 1940 and 1941 a recurrence of her attacks of angina prevented her going. She had several attacks of upper respiratory infections and on at least two occasions in the last four years of her life definite bronchopneumonia developed.

During the summer of 1941 she showed rather marked peripheral edema and was treated with digitalis and mercurial diuretics with improvement. In the following winter she fell and fractured her right humerus but recovered without untoward incident.

During the fall of 1943 she became definitely weaker and spent much of her time in bed. Anginal attacks developed while she was lying in bed. Finally on the last day of December 1943 she became much weaker, there was slight fever and she was confused and disoriented. A definite diagnosis of bronchopneumonia was made and on January 8, 1943, when she was within two months of being 92 years old, she died.

Postmortem Examinations

Dr. Lester S. Round performed the necropsy and reported the following diagnoses:

Primary lesions: Bronchopneumonia and interstitial pneumonia.
Secondary or terminal lesions: None
Historical landmarks:
- Atrophy and fibrosis of thyroid gland (marked).
- Arteriosclerosis—generalized.
  - Aneurysm of abdominal aorta.
  - Coronary sclerosis.
  - Arteriosclerosis and arteriolarsclerosis of the kidneys.
- Chronic fibrous pericarditis.
- Chronic fibrous perisplenitis.
- Chronic fibrosing pneumonitis.
- Infarcts of spleen and kidney.
- Chronic cystitis.
- Senile genitalia.

The detailed descriptions of examination of the organs is omitted except the following:

"*Heart:* The heart weighs 450 grams. There is some left-sided hypertrophy. The anterior and posterior descending branches of the left coronary artery show marked beading and thickening with calcification. The lumen is, in places, reduced to a very small caliber, but everywhere patent. This sclerotic condition also applies to the right coronary, but to a lesser degree. The thickness of the myocardial wall

of the two ventricles is not abnormal. The color is somewhat more brown than usual. The consistency is of normal firmness. There are no infarctions or gross evidence of scarring.

"Lungs: The right lung weighs 455 grams; the left, 285 grams. The bronchial mucosae are hemorrhagic and covered with yellowish gray purulent mucus. The right upper lobe shows a confluent bronchopneumonia. No pneumonic process is suspected in the other lobes. The blood vessels are not abnormal.

"Aorta: The aorta is very large and tortuous in the thoracic portion. In the ascending portion and in the arch there is no atheroma. The descending portion shows marked ulceration with papillary and shaggy masses of fibrin overlying ulcers from which a creamy necrotic substance can be squeezed. Over the sacral prominence is a saccular aneurysm 10 cm. long and 7 cm. wide. The wall is greatly thickened and the intima contains considerable adherent fibrin and some calcification.

"Thyroid: In the position of the thyroid is some soft whitish tissue that maintains the outline and shape of the thyroid. It appears to be entirely loose fibrous tissue and fat. Grossly no thyroid tissue is demonstrated. A brownish body, 0.5 cm. in diameter and thought to be a parathyroid gland, is attached to the left lower pole of this tissue."

Microscopic examination of the thyroid is as follows:

"Scattered through the ten sections is an occasional dilated acinus lined by a single layer of slightly flattened cuboidal cells. The lumina contain a pinkish staining homogeneous substance consistent with a colloid. Scattered through some of the sections are small islands of what appears to be parathyroid tissue."

Comment

The chief interest in this record arises from the fact that the patient was certainly one of the earliest patients with severe myxedema who received thyroid, that the subsequent duration of life on thyroid treatment was probably the longest yet recorded, and that she lived normally many years beyond her expectation of life in spite of the fact that during at least the last 20 years she suffered from marked hypertensive and arteriosclerotic heart disease.

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RUPTURE OF AN ANEURYSM OF A SINUS OF VALSALVA INTO THE RIGHT AURICLE*

By Isaac A. Kawasaki, Captain, M.C., A.U.S., and A. S. Benenson, Major, M.C., U.S.A.

Aneurysms of the sinuses of Valsalva have been reported quite rarely; the rupture of one into the right auricle has been reported in only a few instances.

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Maude E. Abbott reported such a case in 1919.[1] One was reported by Laederich and Poumeau-Delille in 1928.[2] Wright [3] in 1937 cited three cases in the literature other than those mentioned above and reported a fourth case. In most instances death resulted after a period of congestive heart failure.

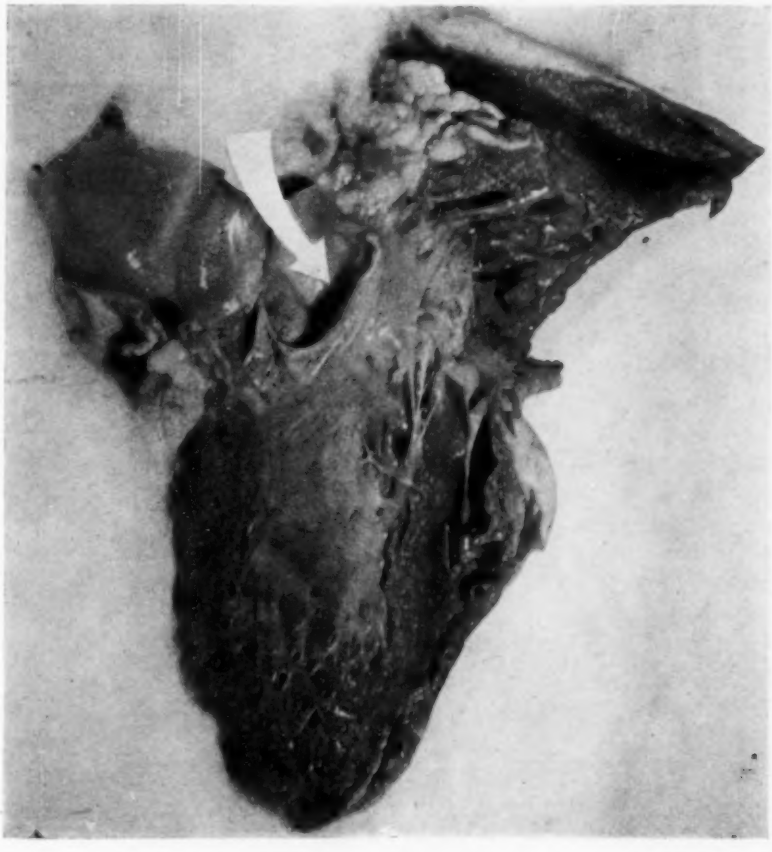

FIG. 1. The arrow points to the opening into the aneurysm in the base of the dilated posterior sinus of Valsalva.

The rarity of the condition justifies reporting this case, an apparently healthy soldier who died within a few minutes of his first complaint, owing to the rupture of an aneurysm of a sinus of Valsalva into the right auricle.

CASE REPORT

This 22 year old soldier had had about 1½ years of military service with no physical disability. He had survived the stress of an unexpected enemy bombardment with no untoward manifestations. One day, about four months thereafter, he came off guard duty and went swimming. One-half hour later he got out of the pool and complained of precordial pain. A few minutes later, he was sitting quietly with body bent forward at the edge of the pool and was seen to roll forward into the water. The

body was removed from the water and artificial respiration was administered unsuccessfully. Death occurred within a few minutes after the first complaint.

Autopsy revealed a well developed and well nourished white male who appeared to be his stated age. Significant findings include diffuse congestion of the lungs, with a weight of 460 and 540 gm. for the right and left lungs respectively. On section, the parenchymatous tissue was congested, purplish red in color, with marked engorgement of the venous tract. Microscopically an enormous amount of congestion was evident with all vessels packed with blood cells. A considerable amount of blood pigment was

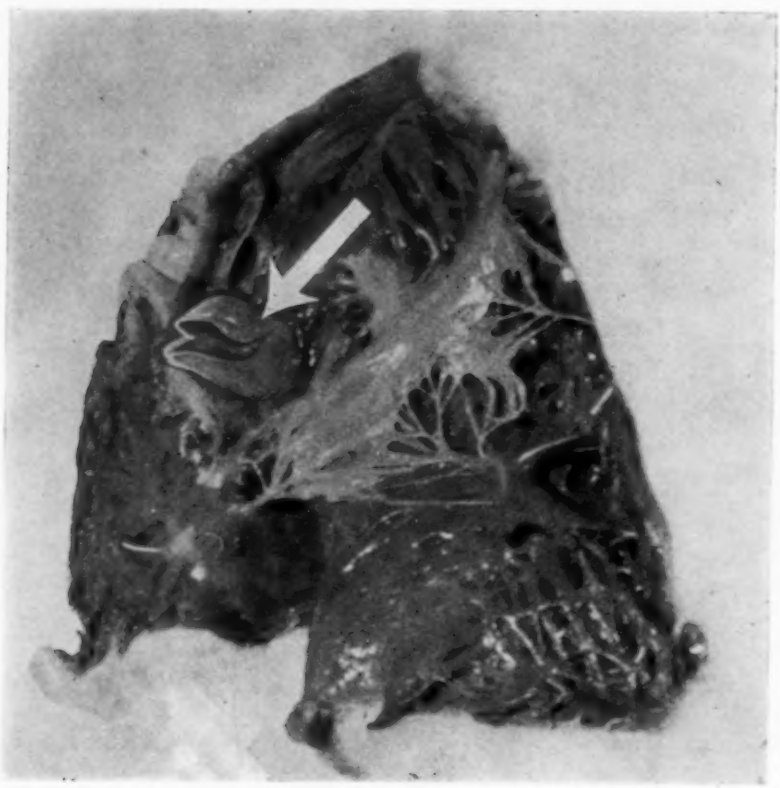

FIG. 2. Aneurysm of the sinus of Valsalva projecting into the right auricle just above the tricuspid valve with a fish-mouth rupture at the apex of the aneurysm.

present and the tissue was edematous. There was marked congestion of the liver. There was congestion of the kidneys, more marked in the medullary zone.

The cause of death was found on examination of the heart. The pericardial sac contained about 75 c.c. of clear straw-colored fluid. The epicardium presented marked engorgement of the venous and capillary network just beneath the serosa, especially marked at the apex of the heart and along the course of the main coronary vessels. The right side of the heart was markedly dilated and very flabby. On section of the myocardium, there were dark red, small, flame-like areas sharply contrasted with the rest of the muscle tissue in the upper portion of the interventricular septum near the junction of the auricular and ventricular walls. The muscle tissue appeared to be swollen. There was no evidence of rupture of any vessels. The coronary tree was

smooth throughout, but the smaller branches within the myocardium stood out more prominently than usual, especially near the involved myocardial area. Here several branches were pale and yellowish with marked pallor within the forks of these branches. The endocardium as a whole was not unusual. There was slight thickening of one of the valve leaflets of the mitral valve. The chordae tendineae were not shortened. The columnae carneae showed no evidence of scarring or thickening.

The aortic valve, at first glance, did not appear especially remarkable. The valve leaflets between the orifices of the two coronary arteries were adherent to form a single septum with a fenestrum so that there was a direct communication between these two sinuses of Valsalva. The left leaflet was much smaller than the other two leaflets. On exposing the posterior sinus of Valsalva, it was found much dilated, measuring 27 by 17 mm. with the valve opened up, and 10 mm. deep (figure 1). At the base of this sinus, there was an opening 12 mm. in diameter into an aneurysmal sac which projected into the right auricle for a distance of 2 cm. The lower margin of this sac was 3 mm. above the edge of the tricuspid valve. It was shaped like the tip of a finger cot, and had, at its apex, a fish-mouth opening extending about half way to the neck of the sac and 5 mm. across at the tip (figure 2). The aorta, above this point, was smooth and glistening, with the usual elasticity and was lined by smooth intima. The foramen ovale was completely closed.

Microscopic sections of the myocardium showed definite edema of the muscle fibers, with some foci of round cell infiltration. There was a definite paucity of red blood cells in the tissue. The aorta above the lesion was microscopically normal, and section through the aneurysmal sac showed it to be constructed of relatively acellular dense connective tissue in which no vascular elements were discerned. There were few elastic tissue fibers evident on special staining, and no evidence of inflammatory reaction.

The character of the lesion was such as to justify the opinion that it was of congenital origin.[1] The exact mechanism of death is conjecturable, but there was anatomical evidence of dilatation of the right heart and myocardial ischemia. The perforated aneurysm constituted a massive arteriovenous fistula with sudden onset. This would result in acute coronary insufficiency, and the change in the myocardium is corroborative evidence of ineffective coronary circulation. It cannot be definitely concluded that this was the cause of death for there were two other possible mechanisms. He might have developed so-called "internal tamponade" from the high pressure stream flowing from the aorta. Secondly, he might have died of cardiac shock through propulsive failure.

Summary

A case of rupture of an aneurysm of the posterior sinus of Valsalva into the right auricle is reported. It resulted in sudden death in an apparently healthy soldier.

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THE INFLUENCE OF OVARIAN ACTIVITY AND ADMINISTERED ESTROGENS UPON DIABETES MELLITUS: CASE REPORT *

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MUCH time and effort have been spent in the study of the probably complex interrelationship existing between the anterior pituitary and the pancreatic islets in their regulation of carbohydrate metabolism. Many points are still obscure. However, there is evidence for an over-all antagonistic action between the hypophysis and the islets of Langerhans which is well supported by the following data.

Hypophysectomy results in a marked improvement in the diabetic state of depancreatized animals,[1, 2] and permanent diabetes can be induced by injecting extracts from the anterior pituitary.[3, 4, 5, 6, 7] These facts indicate that a diabetogenic principle is formed in the pituitary which in excessive amounts, or under certain conditions, proves capable of producing diabetes mellitus through a direct or indirect effect upon the pancreas. This substance probably is produced in connection with the growth hormone of the pituitary and appears to be subject to similar influences.[4, 7, 8]

The administration of sex hormones in large doses is known to exert a suppressive action upon the gonadotropic hormone of the anterior pituitary,[9, 10, 11, 12] as well as upon the growth,[9, 11, 12] the lactogenic,[13, 14, 15, 16, 17] the thyrotropic [11, 12, 19] and the diabetogenic [5, 8, 9, 11, 12, 18] incretions. The action of the sex hormones, particularly the estrogens, upon the diabetogenic principle of the anterior pituitary is less well understood, although some mooted points have already been clarified.

The first attempts to influence favorably the diabetic state by estrogens,[20, 21, 23, 24] were disappointing. It should be noted, however, that the estrogenic preparations were not as potent as later forms and the dosage was comparatively small. For instance, Collens and his co-workers [23] failed to influence the diabetic state with daily doses of estrogenic substances ranging from 100 to 400 R. U. They concluded that estrogens had no effect on human diabetes. Some later workers [22, 25, 26, 27, 28] also found estrogens had no alleviating effect or that in small doses they were actually diabetogenic. Young [7] reported that the administration of estrogens to dogs made permanently diabetic with pituitary extract, failed to modify the diabetes.

On the other hand, evidence has slowly accumulated which shows that diabetes is favorably affected by estrogens in sufficient dosage. Barnes, Regan and Nelson [8] were able to suppress the diabetogenic principle in depancreatized dogs with large doses of estrogens and were able to reduce and control the glycosuria. Nelson and Overholser [5] confirmed the estrogenic suppression of the diabetogenic principle in monkeys and the relief to be obtained in diabetes by the use of ovarian follicular hormone following pancreatectomy.

The frequency of the onset or aggravation of diabetes at the menopause and

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its response to estrogenic hormone have been noted by several workers. Mazer and Israel [29] treated 51 menopausal cases including three with diabetes. In each of the three, diabetes was totally controlled without insulin as long as the patient received at least 2000 R. U. of estrogen every fourth day. With smaller doses the hyperglycemia and glycosuria reappeared. The authors emphasized the importance of employing massive doses, such as 10,000 R. U. every fourth day, whenever inhibition of the anterior pituitary activity was an objective.

Gessler and his co-workers [30] treated five diabetic women at the menopause with 10,000 R. U. of estrogen daily. In three cases, two of whom had a simultaneous onset of the menopause and diabetes, and one a diabetes appearing soon after the menopause, there was a significant lowering of the fasting blood sugar. The other two cases which were not affected by estrogens had no connection with the menopause. In four cases in which urinary assays were done, pituitary gonadotropins were present in the urine before and absent after estrogenic treatment was begun. The effectiveness of estrogens in inhibiting at least certain functions of the anterior pituitary was thus clearly shown.

Spiegelman [31] found that the administration of 10,000 R. U. of estrogenic hormone twice a week to nine diabetic women reduced their insulin requirement. This reduction was maintained for three months after the estrogenic treatment was discontinued. Diminution of the insulin requirement was greater and more sustained in the premenopausal group than in the postmenopausal group, possibly because of the greater capacity of the pituitary to respond.

Cantilo [18] used large doses of estrogen and progesterone in the treatment of menopausal and postmenopausal diabetes mellitus. Treatment was individualized in every case according to the patient's response but usually consisted of from 5 to 10 mg. of estrogen (the particular type of material not stated) and 2 to 5 mg. of progesterone three times a week. No insulin was given and an unrestricted diet was allowed. All patients showed exceptional improvement, even though several had previously exhibited glycosuria and ketonuria.

Gitlow and Kurschner [32] treated 15 cases of diabetes beginning at the menopause or of preëxisting diabetes aggravated at the menopause. These workers found that improvement in the diabetes closely paralleled an improvement in the menopausal symptoms. Subjective improvement was always accompanied by a marked reduction or disappearance of the hyperglycemia and the glycosuria. The urine frequently became sugar free.

The case herewith reported is of interest if only to confirm the ability of the clinician to reduce the insulin requirement in diabetes by the administration of estrogens. Primarily, however, it engages our attention for another reason. It appears to be the first demonstration in a human being of the inhibitory action of therapeutically uninfluenced ovarian function upon the diabetogenic principle of the anterior pituitary.

Case Report

S. S., a white female, was first admitted to the Metropolitan Hospital on October 28, 1943 (figure 1, twenty-first day of observation) because of an uncontrolled diabetes. She was then 31 years old, weighed 114 lbs. and was 61½ inches tall.

Her father died at the age of 83, the cause not known to her. Her mother died of carcinoma of the uterus at an unstated age. One sister aged 27 years and one

brother aged 3 years, both died of diabetes mellitus; and one brother and one sister were living and well.

In 1924, at the age of 12, the patient's diabetes was discovered and insulin started. In 1930 menstruation, although scanty, was established at 28 day intervals. In 1931 she was hospitalized for five months for the control of her diabetes. She was discharged with a measured diet and a maintenance dose of 80 units of insulin daily. From 1931 to 1940 she was admitted to a number of hospitals, usually for the control of insulin shock. On investigation of these admissions, it was found that a marked glycosuria and hyperglycemia developed shortly before the menstrual flow and ended abruptly a few days thereafter. In this post-menstrual period, hypoglycemic reactions often appeared without any premonitory symptoms. During her hospital admissions frequent insulin shocks were noted, necessitating administration of orange juice, sugar, or intravenous glucose. These shocks were first thought to be "hysterical attacks," but it was not unusual to note a marked change of fasting blood sugar in a relatively short space of time. In one instance the blood sugar dropped from 444 to 77 mg. per 100 c.c. in a space of several hours.

In 1940 the patient contracted pulmonary tuberculosis, which was arrested following the production of artificial pneumothorax. Her insulin requirement at the time her tuberculosis became quiescent ranged from 70 to 85 units daily. In 1941 she was admitted to the psychopathic ward of a hospital because of confusion following insulin shock. In 1942 she had a therapeutic abortion. The following year (March, 1943) she was readmitted because of an acute onset of lower abdominal pain, vomiting and diarrhea. At operation an endometrial cyst of the right ovary was found. The left ovary was approximately twice the normal size; it contained numerous follicular cysts and a small endometrial cyst. The right ovary and part of the left were removed. During this stay in the hospital the patient was given 75 units of insulin daily. This was distributed as follows: 35 units of protamine zinc and 15 units of unmodified insulin before breakfast, 15 units of unmodified insulin before lunch, and 10 units of unmodified insulin before supper. While in the hospital she had several mild hypoglycemic reactions. Shortly after her discharge early in June, 1943, she was readmitted for carotinemia and metro-menorrhagia. The latter was believed to be endocrine in origin. During a four months' stay in the hospital she received 70 to 80 units of insulin daily before, during, and after menstruation and 35 units daily during the "mid-period." Despite this regime she showed a marked glycosuria with ketosis at or near the time of menstruation and hypoglycemic reactions during the mid-period.

In October, 1943, she was transferred to Metropolitan Hospital. Detailed blood and urine examinations, including liver and renal function tests, revealed no pathological findings other than the diabetes. The same difficulty was experienced in controlling her diabetes as had been noted formerly. Her course is depicted graphically in figure 1. On December 4, 1943 (the fifty-eighth day of observation, figure 1), estrogenic therapy was begun in an attempt to inhibit the diabetogenic principle of the anterior pituitary. The patient received 1 mg. of estradiol dipropionate intramuscularly.* The following day, the glycosuria decreased from 4.8 per cent to 0.4 per cent. The injection was repeated on December 7, 10, 12, and 14. On December 9 the blood sugar was 70 mg. per 100 c.c., and the following morning the daily dose of insulin was reduced from 35 to 25 units. The patient was maintained on this diminished dosage of insulin throughout the following menstrual period from December 15 to 18 inclusive (days 69 to 72, figure 1). A persistently high urinary sugar with occasional acetonuria and a hyperglycemia of 400 mg. per 100 c.c. of blood developed. As in previous menstrual cycles, the blood sugar dropped within a week to 200 mg. and the urine became aglycosuric. During the mid-period from December 24

* The estradiol dipropionate (Dimenformon Dipropionate) was furnished by Dr. Leo Pirk of Roche Organon Inc., whose courtesy is herewith gratefully acknowledged.

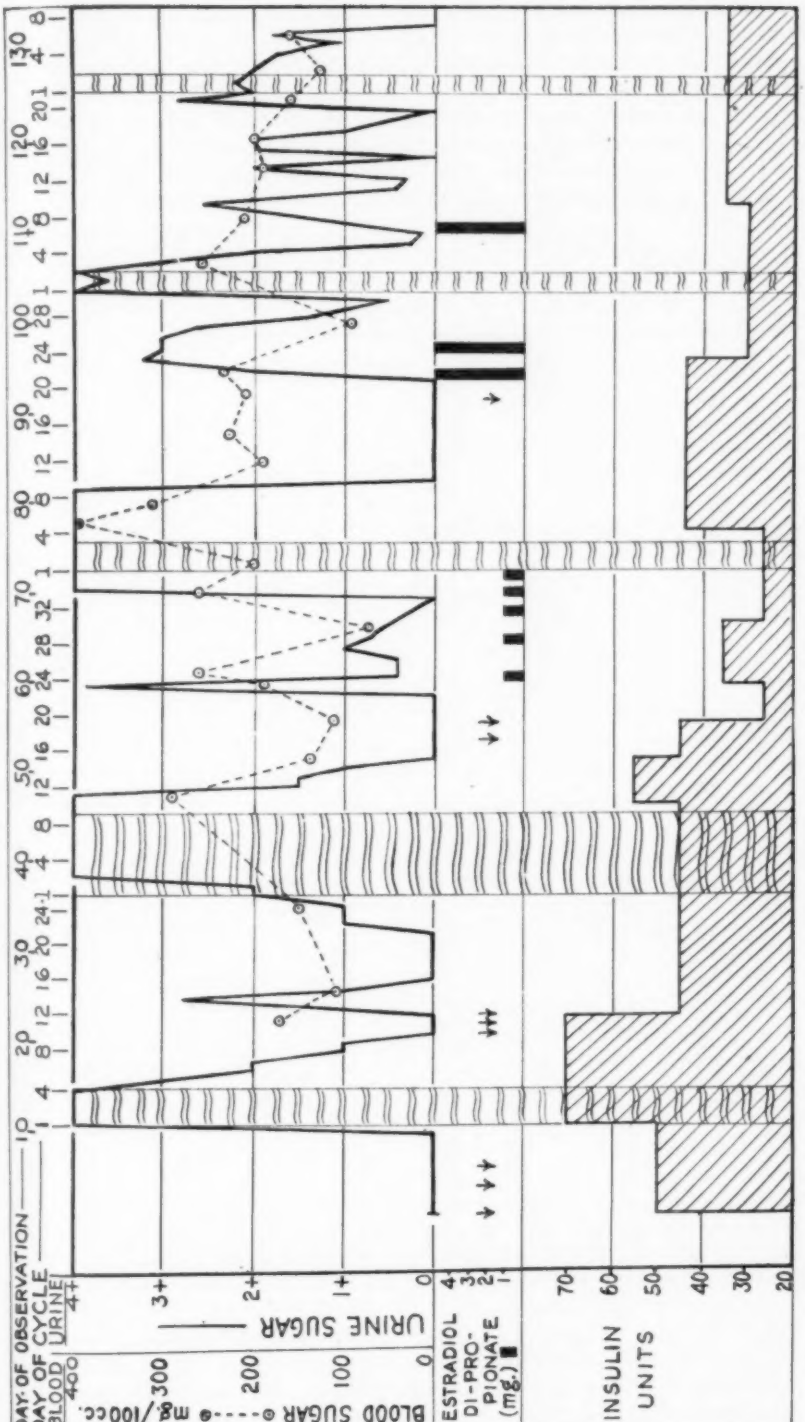

Fig. 1.

Wavy Lined Areas = Menstrual Flow Arrows = Insulin Shock

Glucose in Urine was Determined by Benedict's Qualitative Reagent; 4 Times Daily

1 + = less than 0.5%
2 + = from 0.5 to 1.0%
3 + = from 1.0 to 1.5%
4 + = 2.0% and over

until January 7 (days 78 to 92, figure 1) the fasting blood sugar varied from 200 to 230 mg. per 100 c.c. The urine was free of sugar at all times. A dose of 45 units of insulin daily, begun on December 21, was continued throughout this mid-interval. On January 5 (ninetieth day, figure 1) glycosuria of more than 3 per cent developed. The patient was given 5 mg. of estradiol dipropionate parenterally. The following day the insulin was reduced to 30 units. On January 8 (ninety-third day, figure 1) she again received 5 mg. of estradiol dipropionate; the blood sugar decreased to 95 mg. per 100 c.c. Throughout the menstrual period and for one week following it the

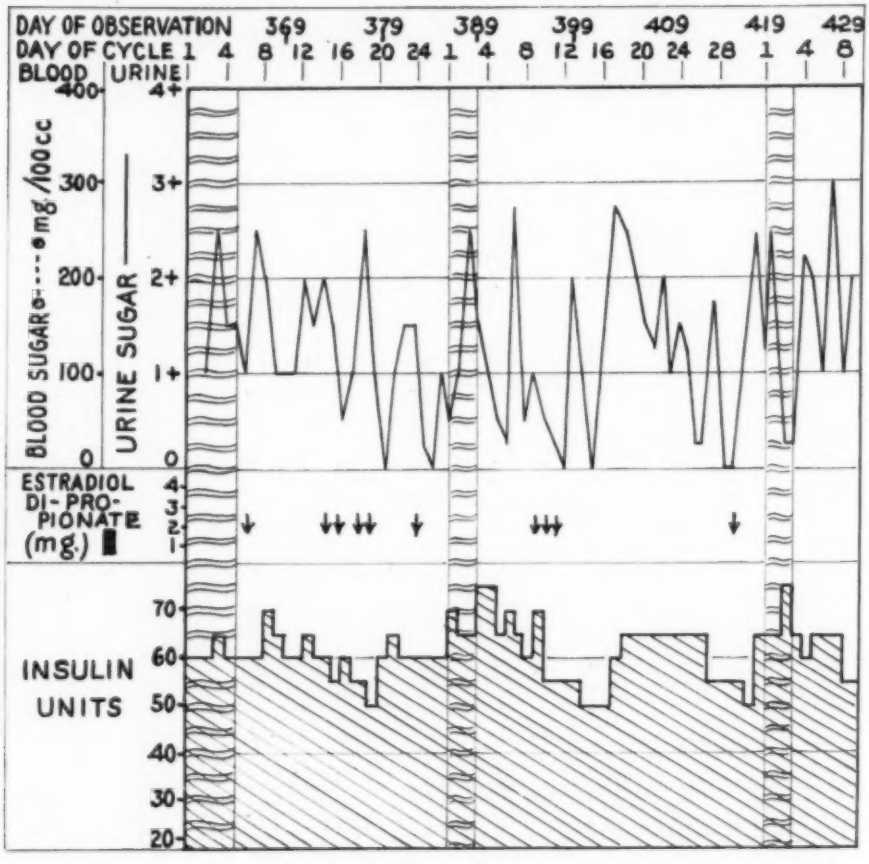

FIG. 2.

insulin dosage was maintained at 25 units daily. Although at times a glycosuria up to 3 per cent appeared, the blood sugar did not rise above 260 mg. per 100 c.c. This was a marked improvement over all previous months. Six days after the onset of her period the patient was given another injection of 5 mg. of estradiol dipropionate and the insulin was increased to and maintained at 35 units daily. Her treatment as noted was continued for the ensuing three weeks. Although there was occasionally some glycosuria, the blood sugar gradually decreased and during the following menstrual period it ranged between 125 and 155 mg. per 100 c.c. She was discharged to the out-patient department of the hospital on February 10, 1944 (one hundred twenty-

sixth day, figure 1). During the entire period of estrogenic therapy there were no "hysterical attacks." Moreover, no sedation was used. The patient stated that subjectively she felt better than she had in years.

The patient was controlled with diet and 35 units of insulin daily for almost two months following her discharge from the hospital, except for periods of 10 to 14 days about the time of menstruation. At such times it was necessary to increase the insulin to 40 units daily.

Following the complete cessation of estrogenic therapy she gradually reverted to her former status (figure 2). In other words, during the mid-period her insulin requirement ranged from 45 to 60 units and during menstruation 10 to 20 additional units were required. When estrogenic therapy was again resumed, the insulin requirement decreased although not as dramatically as before. For the past year the patient has been ambulatory and while mild insulin reactions have occurred, no hospitalization has been necessary.

Discussion

Experimental and clinical evidence points to the fact that large doses of estrogenic hormone have an inhibitory effect on the diabetogenic principle of the anterior pituitary. Conversely, small doses of estrogens either have no effect or are actually diabetogenic. Estrogenic hormone in adequate doses is of greatest value in diabetes when the disease has been initiated or aggravated by an increased liberation of pituitary hormone due to diminished ovarian function. This is particularly true if therapy is instituted before the changes caused by such hormonal imbalance have become irreversible.

How great a part the diabetogenic principle of the pituitary plays in the etiology of human diabetes has not been definitely established, but accumulating evidence accords it an increasingly important rôle. Animal experiments have shown that the diabetes induced by pituitary injections can be made permanent by continuing the injections for a sufficiently long period of time.[3, 7] Does human diabetes become permanent in a similar way? Is that why rapid improvement and easily controlled diabetes usually follow prompt treatment? Lukens and Dohan [33] found that 17 of the 19 patients who had remissions were first seen within four months of the clinical onset of their diabetes. Was this so early that the abnormal changes in the pancreas were still reversible?

The present case is unique in that fluctuations in the patient's own blood and tissue stores of hormone were sufficient to alter her carbohydrate tolerance with every successive menstrual cycle. The temporal relationships of these hormonal changes accord well with the early findings and conclusions of Frank and Goldberger [34] regarding estrogens in circulating and menstrual blood. These workers found an abrupt increase in the amount of female sex hormone in the blood at the mid-interval period, that is between the tenth and fifteenth days. An abrupt decrease was always noted at the onset of flow. Other investigators have made similar observations.[35, 36, 37, 38, 40, 41, 42] It will be noted that our patient has uniformly lost tolerance with the onset of menstrual flow and regained it at mid-interval. The most unusual feature of her entire problem is the degree to which these changes go. They are sufficient to produce coma with menstruation. However, if her diet and insulin are adjusted to prevent such a complication and are not changed again in the middle of the cycle, then hypoglycemic shock invariably occurs.

The important rôle of the variations in estrogenic hormone in the above

alterations is further substantiated by the fact that relatively large doses of estradiol dipropionate have not only improved her glucose tolerance, but have also prevented violent fluctuations in the diabetic status which in the past has led to alternate periods of coma and shock. In other words, during the mid-period of the menstrual cycle when estrogen in the circulating blood is at its peak, the insulin requirement in this patient is much less than near and during the menses when the estrogen level is low. Therefore, her insulin requirement was dramatically decreased by the administration of estrogen. Moreover, the administration of large doses of estrogens has tended to stabilize the diabetic state as shown by the cessation of the previously frequent episodes of hypoglycemia and ketosis.

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39. Smith, G. V., and Smith, O. W.: Quantitative determination of urinary oestrin, Am. Jr. Physiol., 1935, cxii, 340.
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41. Gustavson, R. G., Mason, L. W., Hays, E. E., Wood, T. R., and D'Armour, F. E.: The quantitative determination of estrogenic substances in normal female urine during the menstrual cycle, Am. Jr. Obst. and Gynec., 1938, xxxv, 115.
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EDITORIAL

MURINE TYPHUS FEVER

THIS disease, which clinically resembles a mild form of "classical" typhus fever, seems to have been first recognized as typhus fever by Paullin [1] in Atlanta, Georgia, in 1913. Following his report, similar cases were observed in several other cities along the South Atlantic and Gulf Coasts of the United States. It was not until 1926, however, that Maxcy [2] pointed out clinical and epidemiological differences between these cases and typhus fever as observed in the northeastern United States and in Europe. In 1931 Dyer et al.[3] brought proof that the infectious agent, a Rickettsia, exists naturally in the rat, and that it is conveyed from rat to rat by the rat flea and the rat louse. It is conveyed to man by the rat flea, not by the bite but by rubbing or scratching the infected feces of the flea into the skin.

The clinical features of the disease have been described repeatedly and recently have been reviewed by Stuart and Pullen [4] (in New Orleans) and by Miller and Beeson [5] (in Atlanta). Although the disease is reasonably familiar to physicians where it has been prevalent in the southeastern Seaboard, as it is increasing in frequency and extending northward into areas where it is not well known, a brief description seems appropriate.

The disease, as a rule, occurs sporadically in the endemic areas and is met with chiefly in foodhandlers and others whose occupation brings them into contact with rats. There is probably little difference in susceptibility as regards age, race or sex, except as these factors affect exposure to infection or ease of recognition. The incubation period is usually between eight and 12 days with extremes of five to 15 days.

Although for a few days there may be mild premonitory symptoms such as general aching, slight fever, anorexia and malaise, the onset is characteristically abrupt. There is severe and protracted headache, fever, general malaise, and aching in the muscles. Chills are frequent during the first few days, and gastrointestinal disturbances are common—anorexia, nausea, vomiting, constipation or more rarely diarrhea. Coryza and cough, usually unproductive, occur in about half the cases.

The temperature rises rapidly to a maximum of 103° to 105° F., being sustained during the first week or 10 days, but showing a tendency to partial morning remissions during the second week. The fever usually terminates by a rapid lysis, more rarely by crisis, after two to three weeks.

[1] PAULLIN, J. E.: Typhus fever with a report of cases, South. Med. Jr., 1913, vi, 36.
[2] MAXCY, K. F.: An epidemiological study of endemic typhus (Brill's disease) in southeastern United States, Pub. Health Rep., 1926, xli, 2967.
[3] DYER, R. E., et al.: Experimental transmission of endemic typhus fever of the United States by the rat flea, Pub. Health Rep., 1931, xlvi, 2415.
[4] STUART, B. M., and PULLEN, R. L.: Endemic (murine) typhus fever: Clinical observations of 180 cases, Ann. Int. Med., 1945, xxiii, 520.
[5] MILLER, E. S., and BEESON, P. B.: Murine typhus fever, Medicine, 1946, xxv, 1.

The first and the only highly distinctive feature of the disease is the rash, which occurs in 80 to 90 per cent of white patients, but is often inapparent or overlooked in negroes. It appears usually on the fifth to the eighth day, rarely earlier. It appears first and in greatest profusion on the chest and upper abdomen. It may be sparse and limited to a few spots in this region. Later it spreads, as a rule, involving also the back and proximal segments of the limbs. The palms, soles and face are rarely involved. The rash is composed of small, bright-red macules or slightly elevated papules 2 to 5 mm. in diameter, which fade on pressure. Later they become more dusky, do not fade entirely on pressure, and in severe cases may become petechial. They usually fade after about six days, leaving a brownish discoloration. The eruption may at times be confused with that of typhoid fever, measles, meningococcemia, drug rashes, and particularly tick-borne Rocky Mountain spotted fever. Typically the distribution is different in the latter, starting on the wrists and ankles, then extending to the trunk, and often involving the palms and soles and occasionally the face. It is much more often hemorrhagic. Differentiation of mild cases of Rocky Mountain spotted fever may be quite difficult and require animal inoculation or crossed immunity tests.

Severe manifestations and serious complications are uncommon. Occasionally there may be lethargy or stupor, or confusion and delirium. Bronchopneumonia has been reported in about 5 per cent of the cases. Serious cardiac complications are rare. The mortality is less than 5 per cent.

Physical examination reveals little that is distinctive besides the rash. The spleen is palpable in about 30 per cent of the cases. The leukocyte count is usually about normal, with a tendency to leukopenia during the first week and to a slight leukocytosis during the later period.

After about eight days (five to 20 days) the Weil-Felix reaction (agglutination of the OX_{19} strain of the proteus bacillus) usually becomes positive. A titer less than 1–80 is of no significance, and titers of 1–320 and 1–640 have been observed in conditions other than Rickettsial infections. It often reaches 1–2000 or higher. A similar reaction is given by patients with classical typhus and with Rocky Mountain spotted fever.

A positive diagnosis can usually be reached by intraperitoneal injection into nearly grown male guinea pigs or white rats, of 1 c.c. of blood obtained during the early stage of the disease. After an incubation period of five to nine days the animal shows a fever (104.5° to 104.5° F.) lasting several days and commonly accompanied by erythema and edema of the scrotal skin. Many Rickettsiae can be demonstrated in the cells scraped from the tunica vaginalis. Such scrotal lesions are not produced by other species of Rickettsia, although thromboses and necroses of the scrotal skin may occur after injection of blood from cases of the severe form of Rocky Mountain spotted fever.

A diagnosis can also be made by biopsy of a macule in the skin. The characteristic lesion is swelling and proliferation of the endothelium of the

small vessels. In suitably prepared sections the organisms can be recognized (by experienced observers) in the cytoplasm of the endothelial cells. In Rocky Mountain spotted fever the organisms characteristically invade the nucleus, and the smooth muscle cells of the arterioles are involved.

The disease is caused by *Rickettsia mooseri,* which is closely related to *R. prowazeki,* the cause of classical (European, epidemic, louse-borne) typhus fever. Like the other Rickettsiae, it is a minute coccoid to rod-shaped organism which is not demonstrable by the usual aniline dyes but stains readily by special methods (Castaneda or Giemsa stains). They are not filtrable (except the species *R. burneti,* which causes Q fever), but resemble viruses in growing only within living cells. They can be grown in suitable tissue cultures and in the yolk sac of developing chick embryos. Guinea pigs which have been inoculated with either *R. mooseri* or *R. prowazeki* usually recover and are immune to reinoculation. There is a cross immunity between the two species. The two diseases are differentiated mainly by the difference in epidemiology and the milder clinical course of the endemic type (mortality less than 5 per cent as contrasted with 20 to 80 per cent in epidemic louse-borne typhus). The Rickettsiae can be differentiated by complement fixation tests (Plotz), however, and most readily by animal inoculation. *R. prowazeki* does not cause the scrotal reaction characteristic of *R. mooseri.*

Although many epidemics of louse-borne typhus fever have occurred in the United States following introduction of the infection from abroad, none has been traced to any endemic focus in this country. Sporadic cases of typhus fever, however, have been recognized in the northeastern United States since Brill's description of this form of the disease (1898–1911). As there was little evidence of contagiousness and as the disease was mild and clinically resembled the typhus fever observed in the Southern States, it was assumed that they were identical. Later, however, Zinsser [6] showed that the agent obtained from Brill's disease was identical with that of European (louse-borne) typhus in its pathogenicity and immune reactions. It, therefore, seems unwarranted to apply the term Brill's disease to murine typhus. There is no obvious reason why epidemics might not arise from such cases under suitable conditions, though thus far this has not occurred.

Although murine typhus has been commonly termed endemic, flea-borne typhus, recent observations have shown that it can be conveyed from man to man by the body louse (by the inoculation of the feces or crushed tissue of the louse, not by the bite). When conditions were suitable, with overcrowding and heavy louse infestation, epidemics of murine typhus have occurred (in Mexico).[7]

It is also possible that infection may be acquired without actual contact with fleas. Rickettsiae remain viable in desiccated feces of the flea, and there

[6] ZINSSER, H., and CASTANEDA, M. R.: On the isolation from a case of Brill's disease of a typhus strain resembling the European type, New England Jr. Med., 1933, ccix, 815.

[7] ZINSSER, H.: Virus and rickettsial diseases, 1941, Harvard University Press, Cambridge, p. 872.

is some reason to believe that infection may be acquired by inhalation. Rats can also be infected by feeding infectious material. As Rickettsiae have been demonstrated in rat urine, it seems likely that food contaminated by infected rat urine may be a potential source of infection in man.

Brigham and Pickens[8] have reported finding murine typhus in house mice. The practical significance of this is not yet known, but if the infection in mice behaves as it does in rats and becomes wide spread, the possibilities of its transmission to man are obvious.

Although the practical significance of these latter observations is not yet known, it is possible that infection by inhalation or ingestion may have contributed to the recent geographical spread of the disease. Originally limited largely to the coastal cities, it has gradually spread northward, reaching West Virginia, Tennessee, Arkansas and Iowa, and has been found in Cincinnati, Cleveland and Washington, D. C.[9] It promises to become wide spread in this country wherever rats abound. It is widely distributed, particularly in coastal regions, throughout the world.

There is no specific treatment. Rickettsiae are not susceptible to sulfonamides, penicillin or other known forms of chemotherapy. Immune sera have not yielded any convincing benefit. Practically preventive measures are limited largely to reduction of the rat population, particularly rat-proofing of buildings containing food stuffs and continuous efforts at trapping and poisoning. Considerable protection, probably relatively brief, is afforded by suitable vaccine. Practically, however, its use would be limited to those whose occupation exposes then to unusual risk of infection, or to communities if an actual epidemic should arise. Fortunately the mortality has been very low except in those debilitated by malnutrition or chronic disease.

[8] Brigham, G. D., and Pickens, E. G.: Strain of endemic typhus fever virus from house mice (Mus musculus musculus), Pub. Health Rep., 1943, lviii, 135.

[9] Topping, N. H., and Dyer, R. E.: Recent extension of typhus in the United States, Am. Jr. Trop. Med., 1943, xxiii, 37.

REVIEWS

The Diagnosis of Nervous Diseases. By Sir James Purves-Stewart, K.C.M.G., C.B. 880 pages; 22 × 14.5 cm. 1945. Williams & Wilkins Company, Baltimore. Price, $11.00.

This is the latest edition of a standard clinical guide to neurologic and psychiatric conditions. In spite of war time paper shortages, it is well printed on excellent quality paper. It is not a complete textbook in any sense and deals little with the treatment of the conditions it describes. It is well illustrated with many clear and original diagrams of nerve tracts, as well as numerous photographs of clinical conditions. The style is marked by the clarity of writing for which British authors are justly famed.

Revising it in the midst of World War II, the author was able to introduce the description and discussion of many new neurologic syndromes, such as the crush syndrome and the transient blindness of aviators. Included also is much recent material concerning endocrine physiology.

The book is weak in its discussion of psychological and psychiatric conditions. The author gives in detail the technic of giving the Stanford Revision of the Binet Simon intelligence test. Unfortunately the 1916 revision is used and not the 1937 revision which is far more accurate. The discussion of various neuroses is archaic and shows no understanding of dynamic principles or of recent advances made in this field. Aside from these defects the book is a useful and compact reference volume and serves as an excellent guide for the thorough examination of neurologic conditions.

H. W. N.

Thoughts of a Psychiatrist on the War and After. By William Alanson White, M.D. Republished Essay. 28 pages; 26 × 18 cm. 1942. William Alanson White Psychiatric Foundation, Inc. (Originally copyrighted by Paul E. Hoeber in 1919). Price, $1.50.

This is a small but very readable book. The author is one of the most brilliant thinkers and clear writers that American psychiatry has produced. In this book he traces the unconscious and instinctive forces, the mental mechanisms, of social organization and development. The psychological principles back of all conflict, including war, are clearly described. The problems and dangers confronting victor nations and peace-makers are outlined.

Although written during World War I, the principles described are so sound and so universal, it is difficult to believe that the author is not describing World War II. In the sense that the truth is always timely, this book is well worth reading now and throws considerable light on what to expect in the future.

H. W. N.

BOOKS RECEIVED

Books received during May are acknowledged in the following section. As far as practicable, those of special interest will be selected for review later, but it is not possible to discuss all of them.

Human Physiology. Ninth Edition. By E. H. Starling, M.D., F.R.C.P., F.R.S. Edited and revised by C. Lovatt Evans, D.Sc., F.R.C.P. 1155 pages; 24.5 × 16 cm. 1945. Lea & Febiger, Philadelphia. Price, $10.00.

Cornell Conferences on Therapy. Volume 1. Edited by Harry Gold, M.D. 322 pages; 21 × 14 cm. The MacMillan Company, New York. 1946. Price, $3.25.

Agnosia, Apraxia, Aphasia. 2nd Edition Revised. By J. M. NIELSEN, M.D., F.A.C.P., assisted by J. P. FITZGIBBON, M.D. 292 pages; 24 × 16 cm. 1946. Paul B. Hoeber, Inc., New York. Price, $5.00.

Principles and Practice of Tropical Medicine. By L. EVERARD NAPIER, M.D., F.A.C.P., formerly Director of Tropical Medicine, Calcutta. 917 pages; 25.5 × 16.5 cm. 1946. The MacMillan Company, New York. Price, $11.00.

Electrocardiography. Second Edition, Revised. By LOUIS N. KATZ, M.A., M.D., F.A.C.P. 883 pages; 26.5 × 17.5 cm. 1946. Lea & Febiger, Philadelphia. Price, $12.00.

Exercises in Electrocardiographic Interpretation. Second Edition, Revised. By LOUIS N. KATZ, M.A., M.D., F.A.C.P. 288 pages; 26.5 × 17 cm. 1946. Lea & Febiger, Philadelphia. Price, $6.00.

The Diagnosis and Treatment of Pulmonary Tuberculosis. By MOSES J. STONE, M.D., and PAUL DUFAULT, M.D., F.A.C.P., with Foreword by HENRY D. CHADWICK, M.D. 325 pages; 20.5 × 14 cm. 1946. Lea & Febiger, Philadelphia.

Carbohydrate Metabolism. By SAMUEL SOSKIN, M.D., and RACHMIEL LEVINE, M.D. 315 pages; 25 × 17.5 cm. 1946. University of Chicago Press, Chicago. Price, $6.00.

Research and Regional Welfare. Papers Presented at a Conference on Research at the University of North Carolina, Chapel Hill, May, 1945. Edited by ROBERT E. COKER, with foreword by LOUIS R. WILSON. 229 pages; 23.5 × 16 cm. 1946. University of North Carolina Press, Chapel Hill. Price, $3.00.

A History of Medicine. By DOUGLAS GUTHRIE, M.D., F.R.C.S. with Introduction by SAMUEL C. HARVEY, M.D., F.A.C.S. 448 pages; 24 × 16 cm. 1946. J. B. Lippincott Company, Philadelphia, London. Price, $6.00.

New Human Embryology. By BRADLEY M. PATTEN, Professor of Anatomy, University of Michigan Medical School. 776 pages; 26 × 17 cm. 1946. The Blakiston Company, Philadelphia. Price, $7.00.

The Vitamins in Medicine. Second Edition. By FRANKLIN BICKNELL, D.M., M.R.C.P., and FREDERICK PRESCOTT, M.Sc., Ph.D. 916 pages; 23.5 × 16 cm. 1946. Grune & Stratton, Inc., New York. Price, $12.00.

Curare Intocostrin. Introduction by H. SIDNEY NEWCOMER, M.D. 292 pages; 21.5 × 14 cm. 1946. E. R. Squibb & Sons, New York.

Oxford Loose-Leaf Medicine. Supplements. 14 pamphlets. Oxford University Press, New York. 1946.

DAVID P. BARR, A.B., M.D., LL.D., F.A.C.P.

PRESIDENT, AMERICAN COLLEGE OF PHYSICIANS, 1946–47

DAVID PRESWICK BARR

Born, Ithaca, New York, August 23, 1889; A.B., 1911, Cornell University; M.D., 1914, Cornell University Medical College; LL.D., 1929, Central College (Fayette, Mo.); House Officer, 1914–16, Bellevue Hospital; Assistant Physician, 1916–17, and Research Fellow, 1919–22, Russell Sage Institute; First Lieutenant, 1917–19, Medical Reserve Corps, American Expeditionary Forces; Adjunct Assistant Visiting Physician, 1919–22, and Assistant Physician, 1922–24, Bellevue Hospital; Instructor in Medicine, 1916–22, and Assistant Professor of Medicine, 1922–24, Cornell University Medical College; Physician-in-Chief, 1924–41, Barnes Hospital, St. Louis; Busch Professor of Medicine, 1924–41, Washington University Medical College, St. Louis; Physician-in-Chief, 1941 to date, New York Hospital; Professor of Medicine, 1941 to date, Cornell University Medical College; Honorary Consulting Physician, 1942 to date, Bellevue Hospital.

Fellow, and member of the Council on Pharmacy and Chemistry, American Medical Association; Fellow (1927), and member of the Board of Regents for many years, American College of Physicians; President, 1938–39, Association for the Study of Internal Secretions; Diplomate and member, American Board of Internal Medicine; New York State and County Medical Societies; American Society for Clinical Investigation; Central Society for Clinical Research; Association of American Physicians; New York Academy of Medicine; Harvey Society; Practitioners Society of New York; New York Medical and Surgical Society; Committee on Gas Casualties, National Research Council; Committee, War-Time Graduate Medical Meetings; Sigma Nu and Alpha Omega Alpha Fraternities; author of many published papers and editor of "Modern Medical Therapy in General Practice."

Office: 525 E. 68th Street, New York 21, New York.

HUGH J. MORGAN, B.S., M.D., D.Sc., F.A.C.P.

PRESIDENT-ELECT, AMERICAN COLLEGE OF PHYSICIANS, 1946–47

HUGH JACKSON MORGAN

Born, Nashville, Tennessee, January 25, 1893, son of Joseph Bedinger and Jean Gibson Morgan. Married Robert Ray Porter July 22, 1924; children—Caroline Lee, Hugh Jackson, Jean Gibson, and Robert Porter.

Vanderbilt University, B.S., 1914; Johns Hopkins University School of Medicine, M.D., 1918; University of North Carolina, D.Sc. (Hon.), 1946.

Resident house officer, Johns Hopkins Hospital, 1919–20; assistant resident physician, Johns Hopkins Hospital, 1920–21; instructor in medicine, Johns Hopkins University School of Medicine, 1920–21; assistant and resident physician, Rockefeller Institute of Medical Research, 1922–24, traveling fellow (Europe), 1924–25; Associate Professor of Medicine, Vanderbilt University School of Medicine, 1924–28; Professor of Clinical Medicine, 1924–35; Professor of Medicine, Vanderbilt University School of Medicine, and Physician-in-Chief, Vanderbilt University Hospital 1935– . On leave of absence February 1, 1942—January 1, 1946.

Served as private and 1st lieutenant, Medical Corps, U.S. Army, A.E.F., 1917–18; lieutenant colonel, M.R.C., U.S. Army, 1940; colonel, M.C., U.S. Army, 1942; brigadier general, U.S.A., 1942–1946. Chief Consultant in Medicine to The Surgeon General, U.S. Army, 1942–46. D.S.M., 1945.

Fellow, American College of Physicians; Association of American Physicians; Board, Scientific Directors, International Health Division, Rockefeller Foundation; Society for Clinical Investigation; American Clinical and Climatological Association; American Association for the Advancement of Science; American Medical Association; Southern Medical Association; Phi Beta Kappa, Alpha Omega Alpha, Phi Delta Theta, Sigma Xi. Methodist.

Office: Vanderbilt University Hospital, Nashville 4, Tennessee; residence: White Bridge Pike, Nashville 5, Tennessee.

COLLEGE NEWS NOTES

New Life Member

Dr. Leo E. Westcott, F.A.C.P., Kalamazoo, Mich., has become a Life Member of the American College of Physicians, under date of May 27, 1946.

Gifts to the College Library

The following gifts to the College Library are gratefully acknowledged:

Victor W. Bergstrom, F.A.C.P., Binghamton, N. Y.—2 reprints
Nathan Blumberg, F.A.C.P., Philadelphia, Pa.—1 reprint
Mortimer J. Cantor, (Associate), Brooklyn, N. Y.—1 reprint
Oscar G. Costa-Mandry, F.A.C.P., San Juan, P. R.—2 reprints
C. Wesley Eisele, F.A.C.P., Chicago, Ill.—1 reprint
Norbert Enzer, F.A.C.P., Milwaukee, Wis.—1 reprint
Irving Greenfield, F.A.C.P., New York, N. Y.—1 reprint
Percy G. Hamlin, (Associate), Santa Barbara, Calif.—5 reprints
Chester S. Keefer, F.A.C.P., Boston, Mass.—7 reprints
John E. Leach, F.A.C.P., New York, N. Y.—1 reprint
Arthur G. Lueck, (Associate), Great Lakes, Ill.—1 reprint
Frederick W. Niehaus, F.A.C.P., Omaha, Nebr.—3 reprints
Hilton S. Read, F.A.C.P., Ventnor City, N. J.—3 reprints
Bernard Seligman, F.A.C.P., Brooklyn, N. Y.—3 reprints
Ralph L. Shanno, F.A.C.P., Forty Fort, Pa.—2 reprints
Jacob Jesse Singer, F.A.C.P., Beverly Hills, Calif.—1 reprint
Frederick R. Weedon, (Associate), Jamestown, N. Y.—2 reprints
Burton L. Zohman, F.A.C.P., Brooklyn, N. Y.—5 reprints

Dr. Leland P. Shipp, (Associate), Battle Creek, Mich., has donated the following books to the College Library:

"Lectures on the Principles and Practice of Physic," as delivered at King's College, London, by Thomas Watson, M.D. (Revised, with additions by D. Francis Condie, M.D.), published by Blanchard and Lea, Philadelphia, 1852.

"A Text-book of Practical Medicine," Vol. I, by Felix von Niemeyer, M.D., George H. Humphreys, M.D., and Charles E. Hackley, M.D., published by D. Appleton and Company, New York, 1871.

"A Treatise on the Practice of Medicine," by George B. Wood, M.D., Fifth Edition, Vol. II, published by J. B. Lippincott and Co., Philadelphia, 1858.

Medical Consultants Appointed to Assist in Graduate Training Program, Medical Corps, U. S. Navy

Vice Admiral Ross T. McIntire, (MC), U.S.N., F.A.C.P., Surgeon General of the Navy has announced the appointment of 16 members of the Reserve Consultants Board to the Bureau of Medicine and Surgery. All are outstanding specialists in their respective fields. They will assist the Bureau in furthering the graduate training program, which, in addition to increasing the professional proficiency and improving the standards of medical practice, is designed to afford Naval medical officers the opportunity to train in medical specialties and to qualify for American Board certification, Fellowship in one of the American Colleges, or other marks of distinction in the same manner as doctors engaged in civilian practice.

Among those appointed are the following:

Captain F. J. Braceland, (MC), U.S.N.R., F.A.C.P., Secretary, American Board of Psychiatry and Neurology.

Commodore Alphonse McMahon, (MC), U.S.N.R., F.A.C.P., Associate Professor of Medicine, St. Louis University School of Medicine.

Dr. J. Roscoe Miller, F.A.C.P., Dean and Associate Professor of Medicine, Northwestern University School of Medicine.

1947, Annual Session, American College of Physicians, Chicago, April 28–May 2.

The 28th Annual Session of the College will be held at the Palmer House, Chicago, Ill., from Monday through Friday, April 28–May 2, 1947. Dr. LeRoy H. Sloan, Chicago, will be General Chairman, in charge of local arrangements. He has already started the appointment of committees and an enthusiastic group are working on the preparation. Dr. Sloan will be responsible, not only for local arrangements, but for the program of clinics and panel discussions. Both of these programs were inadequate to accommodate the large numbers attending the Annual Sessions in Philadelphia in 1946. Therefore, adequate extension of these programs is being planned. Dr. David P. Barr, of New York, President of the College, is preparing the program of General Sessions and Morning Lectures. Watch these columns for further announcements during the months to come.

The American College of Physicians Committee on Nominations 1946–1947

In accordance with provisions of the By-Laws, President David P. Barr, on May 21, 1946, appointed the following Committee on Nominations, for 1946–1947, whose duties shall be to nominate candidates for elective offices and for the Board of Regents and Board of Governors. The nominations for the elective officers will be published at least one month in advance of the Annual Business Meeting in 1947, at Chicago. Nominations of Regents and Governors are presented at the Business Meeting without prior publication, but no nominations by this Committee preclude nominations that may be made from the floor of the Meeting:

James J. Waring (Regent), Denver—Chairman
George F. Strong (Regent), Vancouver
Ralph Kinsella (Governor), St. Louis
Asa L. Lincoln (Governor), New York
Jonathan Meakins (Fellow-at-large), Montreal

Reports on Recent A.C.P. Postgraduate Courses

Course No. 5

Course No. 5 Metabolism and Nutrition, was conducted at the Nutrition Clinic of the Hillman Hospital, Birmingham, Ala., June 3–8, 1946, under the Directorship of Dr. Tom D. Spies, F.A.C.P. This was the first course the College had ever sponsored in this particular field. The class purposely was limited in size to twelve registrants. An average of four patients per hour were shown during the clinical sessions. During the teaching sessions minute details were entered into, with explanations of their methods of diagnosing nutritional deficiency diseases and proper therapeutic measures. Question and answer periods were utilized to great advantage. Two afternoons were devoted to field studies, giving the doctors an opportunity to study patients in their own homes and to observe the economic conditions under which they lived. To see the patients in their home surroundings did much to establish a

personal interest in the patients who were shown in the clinical demonstrations. Many of the registrants had never been to Alabama before and had little appreciation of the plight of the South's poor. The patients are already accustomed to the Hillman Clinic social workers coming to their homes, and, therefore, there was no difficulty in having the registrants examine all members of the family, right in the home.

This week of intensive clinical instruction presented a unique opportunity to learn how to cope with the problem of nutritive failure.

Course No. 10

Course No. 10, Internal Medicine, was conducted June 17–28, 1946, at the University of California Medical School and Medical Center, San Francisco, under the Directorship of Dr. Stacy R. Mettier, F.A.C.P. The course was purposely arranged for the two weeks preceding the Annual Meeting of the American Medical Association, thus to give an opportunity to physicians, not only to take the course, but to remain for the A.M.A. Meeting. One hundred and thirteen were registered in the course. At the time of preparation of this news item, the course is just getting under way and details are not ready for publication. The College is gratified with the exceedingly great interest displayed in the course and the fact that College courses are being so well established along the West Coast. The registrants are from all parts of the United States and Canada, showing the importance that they have attached to this exceedingly fine course given by outstanding teachers from the University of California.

Index and Summary of Registrations, Spring Courses, 1946

No.	Title	Institution	Director	Dates
1-A.	Clinical Allergy	Massachusetts General Hospital, Boston, Mass.	Dr. Francis R. Rackemann	March 4–9.
1-B.	Clinical Allergy	Massachusetts General Hospital, Boston, Mass.	Dr. Francis R. Rackemann	April 8–13.
1-C.	Clinical Allergy	Massachusetts General Hospital, Boston, Mass.	Dr. Francis R. Rackemann	July 8–13.
2.	General Medicine	Jefferson Medical College, Philadelphia, Pa.	Dr. Hobart A. Reimann	March 18–23.
3.	General Medicine	University of Texas, School of Medicine, Galveston, Tex.	Dr. Charles T. Stone	March 25–30.
4.	Internal Medicine	Massachusetts General Hospital, Boston, Mass.	Dr. James H. Means	April 1–19.
5.	Metabolism and Nutrition	Nutrition Clinic, Hillman Hospital, Birmingham, Ala.	Dr. Tom D. Spies	June 3–8.
6.	General Medicine	Emory University, School of Medicine, Atlanta, Ga.	Dr. James E. Paullin	April 22–27.
7.	Gastro-enterology	Graduate Hospital, Philadelphia, Pa.	Dr. Henry L. Bockus	April 29–May 4.
8.	Cardiology	Philadelphia General Hospital and the Woman's College of Pennsylvania, Philadelphia, Pa.	Dr. William G. Leaman	May 6–11.
9.	Thoracic Diseases	Department of Postgraduate Medicine, University of Michigan Medical School, Ann Arbor, Mich.	Dr. John Alexander	May 6–11.
10.	Internal Medicine	University of California, Medical School and Medical Center, San Francisco, Calif.	Dr. Stacy R. Mettier	June 17–28.

No.	Fellows	Associates	Non-members	TOTAL	Army	Navy	U.S.P.H.S.	Civilians
1-A.	4	2	0	6	1	0	0	5
1-B.	4	2	0	6	0	0	0	6
1-C.	4	2	0	6	1	1	0	4
2.	35	14	52	101	35	8	6	52
3.	9	7	38	54	9	24	3	18
4.	50	21	11	82	25	4	2	51
5.	4	2	6	12	1	0	3	8
6.	13	7	5	25	7	1	0	17
7.	50	19	9	78	7	3	1	67
8.	113	38	8	159	16	9	0	134
9.	12	15	15	42	7	3	1	31
10.	36	22	55	113	33	11	2	67
	334	151	199	684	142	64	18	460

A. C. P. Postgraduate Courses, Autumn, 1946

(Tentative; not yet final)

The following schedule is still tentative, owing to a few factors requiring further adjustment. At this date (June 19, 1946) the schedule appears to be reasonably final, but some changes have been made recently. Three courses previously announced for the Autumn have been deferred until the Spring of 1947: to wit, a two weeks' course in Internal Medicine, with some emphasis on metabolic disorders, under the direction of Dr. M. A. Blankenhorn, University of Cincinnati; a one week course in Physical Medicine, under the direction of Dr. George Morris Piersol, University of Pennsylvania, Philadelphia; a one week course in Cardiology under Dr. J. Roscoe Miller, Northwestern University, Chicago; a one week course in Tissue Growth and Tumors under Dr. Stanley Reimann at the Lankenau Hospital, Philadelphia.

Dates	Title	Director	Location
Sept. 2–14	Internal Medicine	Dr. R. R. Snowden	Pittsburgh, Pa.
" 23–28	Psychosomatic Medicine	Dr. Franklin Ebaugh	Denver, Colo.
Oct. 7–19	Internal Medicine	Dr. Homer Rush	Portland, Ore.
" 14–18	Clinical Neurology	Dr. Bernard Alpers	Philadelphia, Pa.
" 21–26	Hematology	Dr. Charles A. Doan	Columbus, Ohio
" 21 to Nov. 1	Internal Medicine	Dr. Wallace M. Yater	Washington, D. C.
Nov. 4–9	Allergy	Dr. Robert A. Cooke	New York, N. Y.
" 4–9	Cardiology	Dr. Paul White	Boston, Mass.
" 11–16	Gastro-Enterology	Dr. Walter L. Palmer	Chicago, Ill.
" 18–23	Internal Medicine	Dr. Joseph Hayman	Cleveland, Ohio
" 25 to Dec. 6	Internal Medicine	Dr. J. C. Meakins	Montreal, Que.
Dec. 2–7	Chemotherapy	Dr. W. Barry Wood, Jr.	St. Louis, Mo.
" 2–7	Cardiology	Dr. Frank Wilson	Ann Arbor, Mich.

Detailed bulletins of each course and registration forms will be mailed to all members of the American College of Physicians on or before August 1; also to non-member physicians who have requested being placed on the mailing list.

Fees: (1) Courses in which the maximal registration is restricted to 15 or less, $40.00 per week to members; $80.00 per week to non-members. (2) Courses in which the maximal registration permits more than 15 registrants, $20.00 per week to members; $40.00 per week to non-members.

Inquiries should be addressed to E. R. Loveland, Executive Secretary, American College of Physicians, 4200 Pine St., Philadelphia 4, Pa.

Dr. Hugh J. Morgan, F.A.C.P., President-Elect of the College, Nashville, Tenn., was recently awarded the Distinguished Service Medal. His citation was as follows, "as chief consultant in medicine, Office of the Surgeon General, from February, 1942, to August, 1945, he performed exceptionally meritorious service. Through his untiring efforts the best medical talent in the country was mobilized for service with the Army. Demonstrating marked ability, initiative and judgment in formulating professional policies in the field of medicine and in the assignment of the highest type medical personnel as consultants and chiefs of services, he contributed to the excellent type of medical care received by seriously sick enlisted men and officers. He implemented the procurement of the latest and best supplies and equipment obtainable in order to further alleviate suffering."

Dr. Rafael Rodriguez-Molina, F.A.C.P., San Juan, P. R., was awarded recently the Army Commendation Ribbon, his citation reading as follows: "The Army Commendation Ribbon is hereby awarded for commendable service from May 22, 1942 to February 12, 1946, as assistant chief and chief of the medical service, 161st General Hospital, A.P.O. 851, U. S. Army. During this period Major Rodriguez-Molina displayed outstanding efficiency and devotion to duty. His professional skill coupled with exceptional tact and keen knowledge of the psychology and customs of Puerto Ricans contributed greatly to the accomplishment of the medical mission of this department. His accomplishments reflect great credit on him and on the military service."

Col. Harold C. Lueth, (MC), AUS, F.A.C.P., has received the Army Commendation Ribbon, his citation reading as follows: "During World War II the Medical Department carried out its mission with outstanding success. This achievement was made possible only through the combined efforts of all Medical Department personnel. Your service with the Medical Department has been exceptional when compared with others of the same grade of similar position, and I wish to commend you for your outstanding contribution as liaison officer between the Office of the Surgeon General and the American Medical Association from March 15, 1942 to February 25, 1945."

Lieut. Col. Charles C. Verstandig, (MC), AUS, (Associate), New Haven, Conn., was recently awarded the Army Commendation Ribbon. His citation was as follows: "During World War II the Medical Department carried out its mission with outstanding success. This achievement was made possible only through the combined efforts of all Medical Department personnel. Your service with the Medical Department has been exceptional when compared with others of the same grade of similar position, and I wish to commend you for your outstanding contribution as medical director, Armed Forces Recruiting and Induction Station, New Haven, from March 1, 1943 to December 31, 1945."

Col. Cleve C. Odom, (MC), USA, F.A.C.P., has been awarded the Legion of Merit. His citation was as follows: "Colonel Cleve C. Odom, Medical Corps, Army of the United States, while serving as Commanding Officer of Mason General Hospital, distinguished himself through outstanding service. Colonel Odom expanded Mason General Hospital from a 1,320 to a 3,032 bed hospital during his tenure of command and provided instruction of the highest quality for medical officers and nurses undergoing instruction in the School of Military Neuropsychiatry operated at this station. Through his broad experience in neuropsychiatry and hospital administration, untiring efforts, remarkable initiative and enthusiastic and virile leadership, Mason General

Hospital attained a prominent place in military neuropsychiatry and administered the best of care and treatment to the neuropsychiatric patients of the Army. His cumulative achievements reflect great credit on himself and the medical corps."

Surplus Army Hospitals Released to Veterans Administration

The Army's great general hospitals, built to the latest medical and surgical standards for the care and treatment of its wounded and sick during the war, are being released as rapidly as the decrease in the patient load justifies and offered first to the Veterans Administration for its rapidly expanding program for medical care for veterans.

The transfers have been made as part of the Army's comprehensive plan, devised before hostilities had ceased, to effect a smooth transition when responsibilities for the care of the sick and wounded were transferred from the Army to the Veterans Administration.

The War Department program is being carried out through close coöperation between Major General Norman T. Kirk, F.A.C.P., The Surgeon General, and Dr. Paul R. Hawley. F.A.C.P., Medical Director of the Veterans Administration, who before retirement from the Army as a Major General, was Chief Surgeon in the European Theatre of Operations.

Already 11 hospitals have been transferred completely to the Veterans Administration, according to a report from Surgeon General Kirk.

Rheumatic Fever Council Receives Initial Fund of $50,000

At Atlantic City on May 29, it was announced that the American Legion had presented the American Council on Rheumatic Fever with a check for $50,000 to start a nation-wide campaign to raise the minimum of $5,000,000 a year to combat this disease. The presentation was made by Dr. Leonard G. Rowntree, F.A.C.P., Philadelphia, who is chief medical adviser to the American Legion. The gift was accepted by Dr. Roy W. Scott, F.A.C.P., Cleveland, who is President of the American Heart Association, which was the organizer of this Council on Rheumatic Fever. The American College of Physicians has donated $1,000 to the Fund.

New Jersey Members Hold Meeting at Atlantic City, May 22

Fellows and Associates of the American College of Physicians of New Jersey held a luncheon meeting at the Hotel Traymore, Atlantic City, May 22, during the Annual meeting of the New Jersey State Medical Society. Arrangements were conducted by Dr. Johannes Pessel, F.A.C.P., Trenton. Dr. George H. Lathrope, F.A.C.P., Governor for New Jersey, presided. Dr. William D. Stroud, F.A.C.P., Philadelphia, Treasurer of the College, and Mr. E. R. Loveland, Executive Secretary of the College, addressed the group about College matters. Fifty-four members were in attendance.

Western Michigan Members Hold Second Regional Meeting

Members of the American College of Physicians in Western Michigan, as announced recently in these columns, have organized for the conduct of three or more scientific and social regional meetings each year. The second such meeting was held at the Percy Jones General Hospital, May 1, 1946, with 31 members in attendance. Dr. William N. LeFevre, Muskegon, is the Secretary.

College News from Puerto Rico

During the annual meeting of the Puerto Rico Medical Association held during December 1945, the following members of the American College of Physicians took active part in the meeting:

Dr. Franklin L. Hanger, Jr., F.A.C.P., of Columbia University, New York City.

Dr. Henry L. Bockus, F.A.C.P., of the University of Pennsylvania, Philadelphia.

Dr. Ramón M. Suárez, F.A.C.P., in coöperation with Dr. Tom Spies, F.A.C.P., Birmingham, Ala., talked on the treatment of macrocytic anemia with folic acid.

Dr. Rafael Rodríguez-Molina, F.A.C.P., Major, A.U.S., also took active part in the meeting.

Dr. Federico Hernández, F.A.C.P., of the School of Tropical Medicine was invited by Tulane University to give a short course during a period of two months. Dr. Hernández returned from New Orleans during the month of March.

Dr. O. Costa Mandry, F.A.C.P., of the Health Department of Puerto Rico, was invited by the Rockefeller Foundation to coöperate in the establishment of a public health laboratory in the Dominican Republic. During the second week of January Dr. Costa Mandry visited the Dominican Republic to outline plans for the establishment of this laboratory.

During the month of November the members of the College in Puerto Rico gave an informal luncheon to Dr. R. Rodríguez-Molina on account of his promotion in the Army from Captain to Major.

Dr. Luis M. Morales, F.A.C.P., of San Juan, was appointed during the month of December President of the Puerto Rico Medical Association.

Dr. Ramón M. Suárez, College Governor of the District of Puerto Rico, left at the beginning of April for the Annual Session of the American College of Physicians at Philadelphia to present a paper on the treatment of sprue by folic acid.

Dr. C. C. Carpenter, Dean of The Bowman Gray School of Medicine of Wake Forest College, recently announced the gift of $125,000 from Mr. Bowman Gray, Jr., matching a similar amount recently given to the school by his brother, Mr. Gordon Gray. Both of these gifts are unrestricted in their use.

A department of Preventive Medicine has been organized, and Dr. Thomas T. Mackie of New York has been elected Professor of Preventive Medicine and Chairman of the Division of Medicine.

Dr. John H. Ferguson, Professor of Physiology at the University of North Carolina School of Medicine, addressed the Bowman Gray Medical Society on April 15, 1946. His subject was: "Blood Coagulation and Modern Clinical Applications."

Dr. Edward C. Reifenstein, F.A.C.P., has been honored by the establishment of the Dr. Edward C. Reifenstein Lectureship in Medicine at Syracuse University College of Medicine by the family of Dr. Ellery G. Allen. Dr. Reifenstein became professor emeritus of medicine on July 1, 1940, but has continued as chairman of the department during the war years.

The first lecture was given by Dr. Edgar V. Allen, F.A.C.P., associate professor of medicine at the University of Minnesota, Mayo Foundation, Rochester, April 8, his subject being, "The Challenge of Intravascular Thrombosis."

Dr. Franklin B. Bogart, F.A.C.P., Chattanooga, Tenn., has been elected president-elect of the Tennessee State Medical Association.

Dr. Robert F. Loeb, F.A.C.P., professor of medicine at Columbia University College of Physicians and Surgeons, is one of twenty-nine leaders in American Science recently elected to membership in the National Academy of Science.

Among the speakers at the 180th annual meeting of the Medical Society of New Jersey on May 22, 1946, in the Section of Gastro-enterology and Proctology were Dr. Manfred Kraemer, F.A.C.P., Newark, who spoke on "Intestinal Parasitism in an Army Hospital," and Dr. Louis L. Perkel, F.A.C.P., Jersey City, who spoke on "Esophageal Hiatus Hernia." The officers elected by the section for the coming year were Dr. Louis L. Perkel, F.A.C.P., and Dr. Sigurd W. Johnson, F.A.C.P., Passaic, chairman and secretary, respectively.

The Medical Society of Virginia will hold its annual meeting at Virginia Beach, October 14–16, 1946. Dr. A. B. Hodges, F.A.C.P., will be the chairman of the meeting.

Rear Admiral Kent C. Melhorn, F.A.C.P., (MC), U.S.N., delivered the commencement address before the University of Virginia Department of Medicine on March 20.

Dr. Eugene M. Landis, F.A.C.P., Professor of Physiology at Harvard Medical School, gave the annual Alpha Omega Alpha address, April 19, on "Venous Pressure and Cardiac Failure in the Laboratory and Clinic."

Dr. L. E. January (Associate), was recently separated from the Medical Corps, AUS, as a Lieutenant Colonel, and has been appointed assistant professor of internal medicine at the State University of Iowa.

Dr. George F. Lull, F.A.C.P., Chicago, addressed the first Rocky Mountain Regional Conference on Medical Service and Public Relations at Denver on June 5 on the subject, "Problems of the Returning Medical Officer."

Dr. Arthur C. Christie, F.A.C.P., Washington, D. C., delivered the George W. Holmes annual lecture before the annual meeting of the New England Roentgen Ray Society, at Boston, on May 17, his subject being, "The First Fifty Years of Radiology: The Elements Which Have Contributed to Its Growth as a Great Medical Specialty."

Dr. Stockton Kimball, F.A.C.P., who has been a member of the faculty of the University of Buffalo School of Medicine for many years, was recently appointed dean of the medical school to succeed Dr. Edward W. Koch, deceased.

The forty-seventh annual meeting of the American Therapeutic Society at Atlantic City, May 11–12, 1946, was addressed by the following members of the College:

Dr. Oscar B. Hunter, Jr., F.A.C.P., Washington, D.C., "Correlation of Laboratory Findings with Therapy in Epidemic Hepatitis;"

Dr. David I. Macht, F.A.C.P., Baltimore, Md., "An Experimental Approach to the Therapy of Pemphigus;"

Dr. Daniel L. Sexton, F.A.C.P., St. Louis, Mo., "Thiouracil—Clinical Evaluation Following Two and One Half Years' Experience;"

Dr. Francis M. Pottenger, F.A.C.P., Monrovia, Calif., "Difficulties in Physical Examination of the Chest;"

Dr. David Salkin, F.A.C.P., Hopemont, W. Va., "Postmortem Pneumothorax;"

Dr. Nathan S. Davis, III, F.A.C.P., Chicago, Ill., "The Treatment of Hypertensive Cardiovascular Renal Disease with Ascorbic Acid, Riboflavin and Vitamin B Complex."

Dr. Stanley P. Reimann, F.A.C.P., Philadelphia, Pa., addressed the Ohio State Medical Association at its one hundredth anniversary meeting at Columbus, May 7–9, on "Present Status of Cancer Research."

The Southern Medical Association announces its next annual meeting at Miami, Fla., November 4–7, 1946.

Brigadier General George R. Callender, F.A.C.P., (MC), U.S.A., Commandant of the Medical Department Professional Service Schools, Washington, D. C., was recently awarded the Richard Pearson Strong Paladium Medal of the American Foundation for Tropical Medicine. This medal, including an honorarium of $500, is given by the Winthrop Chemical Company, New York.

Dr. George Albert Gray, F.A.C.P., formerly of Abilene, Tex., retired from the Army with the rank of Lieutenant Colonel recently and has been appointed Director of the San Angelo-Tom Green County Health Unit, San Angelo, Tex.

Dr. Merritt Henry Stiles, F.A.C.P., formerly of Philadelphia, was retired from the Army with the rank of Lieutenant Colonel early this year and is established in practice at 1070 Paulsen Medical-Dental Bldg., Spokane, Wash.

Dr. Herbert Pollack, F.A.C.P., who recently has been serving as Chief Medical Consultant in the European Theater, has retired from the Army with the rank of Colonel and returned to civilian practice at 45 E. 66th St., New York City.

Dr. Hugh R. Leavell, F.A.C.P., for many years Director of Public Health, Louisville, Ky., served from November, 1944, to December, 1945, in the USPHS (R). He has now accepted an appointment as Assistant Director of the Division for the Medical Sciences of the Rockefeller Foundation, New York City, and is assigned to Undergraduate Medical Education in Preventive Medicine.

Dr. Ralph Frederick Schneider (Associate), retired from the United States Naval Reserve during April, is now associated with the Medical Department of the Standard Oil Company, 30 Rockefeller Plaza, New York City. On May 28 he left for South America, where, in conjunction with American Board members in Surgery and Obstetrics, he will participate in a teaching and reorganization program in the hospitals of the Standard Oil Company affiliates.

Dr. Henry B. Gwynn, F.A.C.P., who since 1943 has been Associate Clinical Professor of Medicine at Georgetown University School of Medicine, Washington, D. C., has removed to Mobile, Ala., where he has established an office at 751 Government St.

Dr. George S. Grier, III (Associate), has recently completed a Fellowship in Pathology at the Medical College of Virginia, Richmond, and on July 1 opened his office at 130 26th St., Newport News, Va.

In the May issue of this journal it was inadvertently published that Dr. Marsh McCall, F.A.C.P., had recently retired from the Army with the rank of Lieutenant Colonel, whereas later records disclosed he was separated from duty with the rank of Colonel.

Dr. James E. Paullin, F.A.C.P., Atlanta, presented the dedication address at a meeting of the Fulton County Medical Society, at the Atlanta Academy of Medicine on April 4. It was the occasion of the dedication of the auditorium of the Fulton County Academy of Medicine to Dr. Abner Wellborn Calhoun.

Dr. Joseph C. Edwards, F.A.C.P., Lt. Col., (MC), AUS, with the 21st General Hospital, and now Instructor in Medicine at Washington University, St. Louis, Mo., was awarded the Legion of Merit with a Citation for clinical research during the war, in the Mediterranean and the E. T. O.

Dr. George H. Houck, F.A.C.P., recently retired from the Army of the United States, has accepted an appointment as director of student health, Stanford University.

Dr. Francis M. Pottenger, Sr., F.A.C.P., Monrovia, Calif., has been named to the newly created office of president emeritus of the Los Angeles County Tuberculosis and Health Association. Dr. Pottenger was one of the founders of this Society.

Dr. Cyril M. MacBryde, F.A.C.P., formerly of St. Louis, is now assistant clinical professor of medicine at the University of Southern California School of Medicine, Los Angeles.

The Idaho State Medical Association's annual meeting at Boise, June 17–20, was addressed by Dr. James J. Waring, F.A.C.P., Denver, Colo., on "Various Aspects of Tuberculosis," and by Dr. Ward Darley, Jr., F.A.C.P., Denver, Colo., on "Diagnosis of Rheumatic Fever and Rheumatic Heart Disease."

Dr. Chester S. Keefer, F.A.C.P., Boston, addressed the 92nd session of the Maine Medical Association, at Poland Spring, June 23–25, on streptomycin.

Brigadier General James S. Simmons, (MC), USA, F.A.C.P., Chief of the Preventive Medicine Service, Office of The Surgeon General, was awarded the honorary degree of Doctor of Science from the University of North Carolina, at Chapel Hill, recently.

General Simmons will assume duties as Dean of the Harvard School of Public Health on July 1, when he retires from the Regular Army.

Dr. Harold J. Jeghers, F.A.C.P., Boston, Massachusetts, has been appointed Professor and Director of the Department of Medicine at the Georgetown University School of Medicine and physician-in-chief of the Georgetown University Hospital. The appointment became effective July 1, 1946. Dr. Jeghers has been associated with the Boston University School of Medicine and Boston City Hospital since 1935 and with the Evans Memorial Hospital since 1939.

Dr. Howard Wakefield, F.A.C.P., and Dr. S. W. McArthur, both of Chicago, addressed the Racine County Medical Society, at Racine, Wis., April 18, 1946, on "The Association of Gall Bladder Disease and Heart Disease: Clinical and Experimental Observations."

Dr. Lee D. Cady, F.A.C.P., who formerly served in the Army as a Colonel in the Medical Corps, is now the Branch Medical Director of the U. S. Veterans Administration, representing Texas, Louisiana and Mississippi. He is located in the Mercantile Bank Bldg., Dallas, Tex.

At the Third Annual Meeting of the American Society for Research in Psychosomatic Problems, at Hotel Pennsylvania, New York City, May 11–12, 1946, Dr. Edward Weiss, F.A.C.P., Philadelphia, was elected President, and Dr. William Dock, F.A.C.P., New York City, and Dr. Leonard G. Rowntree, F.A.C.P., of Philadelphia, were elected to the Council.

Dr. Harold G. Trimble, F.A.C.P., was the dinner speaker at the meeting of the Oregon Tuberculosis Association, at Portland, during May.

Dr. Hyman I. Goldstein, Associate, Camden, N. J., addressed the American Association of the History of Medicine at its 19th Annual Meeting, Atlantic City, May 27, 1946, on "The History of Medical Education and of Some Medical Men, in New Jersey."

The American Clinical and Climatological Society will meet at Hershey, Pa., October 21–23, 1946.

Dr. Daniel L. Sexton, F.A.C.P., St. Louis, has been elected Vice President of the Missouri State Medical Association.

Dr. Roy C. Mitchell, F.A.C.P., Mount Airy, has been elected President of the Eighth District County Medical Society of North Carolina.

Retirements from Service

Since the last publication of this journal, the following members of the College have been reported retired or on terminal leave (to June 13, 1946 inclusive).

Horst A. Agerty, Merion Station, Pa. (Capt., MC, AUS)
John J. Archinard, New Orleans, La. (Lt. Col., MC, AUS)
Gerald S. Backenstoe, Emmaus, Pa. (Major, MC, AUS)
George N. Barry, Oklahoma City, Okla. (Lt. Comdr., MC, USNR)
M. Meredith Baumgartner, Janesville, Wis. (Comdr., MC, USNR)
Joseph C. Bell, Louisville, Ky. (Lt. Col., MC, AUS)
Abraham Becker, Detroit, Mich. (Major, MC, AUS)
Lawrence H. Beizer, Rochester, Minn. (Lt. Col., MC, AUS)
Charles A. Bohnengel, New York, N. Y. (Lt. Col., MC, AUS)
Donald W. Bortz, Cleveland, Ohio (Lt., MC, USNR)
Samuel R. Brownstein, New York, N. Y. (Major, MC, AUS)
Harold J. Brumm, St. Joseph, Mo. (Lt. Comdr., MC, USNR)
Samuel Candel, Brooklyn, N. Y. (Comdr., MC, USNR)
Manley J. Capron, Battle Creek, Mich. (Capt., MC, USNR)

Herman M. Chesluk, Detroit, Mich. (Capt., MC, AUS)
George R. Crisler, Winter Park, Fla. (Major, MC, AUS)
Haydn H. Cutler, Houston, Tex. (Lt. Col., MC, AUS)
Mahlon H. Delp, Kansas City, Kan. (Col., MC, AUS)
John S. Denholm, Burlington, N. C. (Col., MC, AUS)
George P. Denny, Boston, Mass. (Col., MC, AUS)
Frederick W. Fitz, Chicago, Ill. (Lt. Col., MC, AUS)
Ralph G. Fleming, Durham, N. C. (Major, MC, AUS)
Robert F. Foster, Seattle, Wash. (Major, MC, AUS)
A. James French, Ann Arbor, Mich. (Lt. Col., MC, AUS)
Franklin W. Fry, Hempstead, N. Y. (Capt., MC, AUS)
Joseph J. Furlong, Milwaukee, Wis. (Lt. Comdr., MC, USNR)
Cleo R. Gatley, Pontiac, Mich. (Major, MC, AUS)
James T. J. Geddis, New York, N. Y. (Major, MC, AUS)
Hermon C. Gordinier, Troy, N. Y. (Lt. Col., MC, AUS)
George A. Gray, Abilene, Tex. (Lt. Col., MC, AUS)
Edward D. Greenwood, Topeka, Kan. (Major, MC, AUS)
William H. Grishaw, Los Angeles, Calif. (Major, MC, AUS)
Morris B. Guthrie, Columbus, Ohio (Col., MC, AUS)
Russell B. Hanford, Spokane, Wash. (Lt. Col., MC, AUS)
Thomas J. Hanlon, St. Louis, Mo. (Major, MC, AUS)
John Harvey, Lexington, Ky. (Lt. Col., MC, AUS)
William R. Hewitt, Tucson, Ariz. (Lt. Col., MC, AUS)
A. Parker Hitchens, Philadelphia, Pa. (Lt. Col., MC, USA)
Edward D. Hoedemaker, Seattle, Wash. (Lt. Comdr., MC, USNR)
A. Gerson Hollander, Brooklyn, N. Y. (Lt. Col., MC, AUS)
Kendall B. Holmes, Fresno, Calif. (Major, MC, AUS)
J. Morris Horn, Fort Worth, Tex. (Major, MC, AUS)
Lewis E. January, Iowa City, Iowa (Lt. Col., MC, AUS)
Henry J. John, Cleveland, Ohio (Lt. Col., MC, AUS)
Louis Krause, Baltimore, Md. (Lt. Col., MC, AUS)
L. Rush Lambert, Fairmont, W. Va. (Col., MC, AUS)
Thomas A. Lebbetter, Yarnouth, N. S., Can. (Col., RCAMC)
Clarence W. LeDoux, Baltimore, Md. (Capt., MC, AUS)
Henry J. Lehnhoff, Jr., Rochester, Minn. (Lt. Col., MC, AUS)
Frederick Lemere, Seattle, Wash. (Lt. Col., MC, AUS)
Victor F. Lief, Far Rockaway, N. Y. (Lt. Col., MC, AUS)
Wallace W. Lindahl, Rochester, Minn. (Major, MC, AUS)
Edward A. Marshall, Cleveland, Ohio (Lt. Col., MC, AUS)
John R. Shannon Mays, Baltimore, Md. (Lt. Col., MC, AUS)
Donald McCarthy, Minneapolis, Minn. (Capt., MC, USNR)
William U. McClenahan, Philadelphia, Pa. (Major, MC, AUS)
Leo J. Meienberg, Portland, Ore. (Lt. Comdr., MC, USNR)
Nathan T. Milliken, Hanover, N. H. (Lt. Col., MC, AUS)
Fred S. Modern, Los Angeles, Calif. (Lt. Comdr., MC, USNR)
Clifford K. Murray, Ventnor, N. J. (Lt. Comdr., MC, USNR)
Alonzo Y. Olsen, Los Angeles, Calif. (Major, MC, AUS)
John R. Osborne, Middletown, N. Y. (Comdr., MC, USNR)
Arthur L. Osterman, Wheeling, W. Va. (Capt., MC, USNR)
Frank Perlman, Portland, Ore. (Lt. Col., MC, AUS)
Herbert Pollack, New York, N. Y. (Col., MC, AUS)
Everett B. Poole, Greenville, S. C. (Col., MC, AUS)
Hans P. Popper, Chicago, Ill. (Major, MC, AUS)
William W. Priddle, Toronto, Ont., Can. (Major, RCAMC)

David E. Quinn, Livermore, Calif. (Col., MC, AUS)
David R. Sacks, San Antonio, Tex. (Lt. Col., MC, AUS)
Charles H. Scheifley, Rochester, Minn. (Capt., MC, AUS)
Sidney Scherlis, Baltimore, Md. (Major, MC, AUS)
Ralph F. Schneider, New York, N. Y. (Lt., MC, USNR)
Leon Schwartz, Philadelphia, Pa. (Surgeon, USPHS (R))
Fred F. Senerchia, Jr., Elizabeth, N. J. (Col., MC, AUS)
Richard M. Shick, Rochester, Minn. (Lt. Comdr., MC, USNR)
James J. Short, New York, N. Y., (Capt., MC, USNR)
Jacob J. Silverman, Staten Island, N. Y. (Major, MC, AUS)
Robert L. Smith, Jr., Rochester, Minn. (Major, MC, AUS)
Dale C. Stahle, Harrisburg, Pa. (Major, MC, AUS)
Edson H. Steele, Los Angeles, Calif. (Comdr., MC, USNR)
Israel Steinberg, New York, N. Y. (Comdr., MC, USNR)
Morris F. Steinberg, New York, N. Y. (Capt., MC, AUS)
Merritt H. Stiles, Philadelphia, Pa. (Lt. Col., MC, AUS)
Stanley R. Szymanski, Livingston, N. Y. (Major, MC, AUS)
Herman Tarnower, Scarsdale, N. Y. (Lt. Col., MC, AUS)
Thomas Van Orden Urmy, Boston, Mass. (Lt. Col., MC, AUS)
Ernest J. Vogel, Arlington, Mass. (Capt., MC, AUS)
Norton W. Voorhies, New Orleans, La. (Capt., MC, AUS)
Joseph C. Watts, Bayside, L. I., N. Y. (Major, MC, AUS)
Edward A. Wilkerson, Houston, Tex. (Lt. Col., MC, AUS)
Ellis W. Willhelmy, La Jolla, Calif. (Lt. Comdr., MC, USNR)
Willis D. Wright, Omaha, Nebr. (Comdr., MC, USNR)
Joseph Ziskind, New Orleans, La. (Major, MC, AUS)

Dr. T. Grier Miller Appointed A. C. P. Marshal

Dr. T. Grier Miller, F.A.C.P., Philadelphia, newly elected Regent of the American College of Physicians, has been appointed by the President, Dr. David P. Barr, as official Convocational Marshal of the College, succeeding Dr. Reginald Fitz, F.A.C.P., Boston, who has resigned.

Fourth International Congress on Tropical Medicine and Malaria to Be Held in the United States

Through the instrumentality of the American Academy of Tropical Medicine plans are under way for the organization of the Fourth International Congress on Tropical Medicine and Malaria to be held in the United States, possibly during 1947, under the official sponsorship of the United States Government, with the collaboration of a number of interested medical and scientific organizations. Already the Southern Medical Association, The American Society of Tropical Medicine, The American Academy of Tropical Medicine, the National Malaria Society, the American College of Physicians and others have adopted resolutions looking toward the organization of such an International Congress and to assist therewith. Dr. Joseph M. Hayman, Jr., F.A.C.P., Professor of Clinical Medicine at Western Reserve University School of Medicine, Cleveland, has been appointed as the official representative of the American College of Physicians in initiating the invitations and making preliminary arrangements.

SPECIAL NOTICE

Announcement is made by Surgeon General Thomas Parran of the U. S. Public Health Service that a grant for the establishment of 125 Fellowships to train physicians and sanitary engineers in public health has just been approved by the National Foundation for Infantile Paralysis.

Each Fellowship provides a year's graduate training in a school of public health or a school of sanitary engineering. The Fellowships will be administered by the Committee on Training of Public Health Personnel, which consists of representatives of schools of public health, the State and Territorial Health Officers, the American Public Health Association and the U. S. Public Health Service.

The Fellowships are available either during the academic year beginning in the fall of 1946 or the fall of 1947, and are open to men and women, citizens of the United States under 45 years of age.

The purpose of the Fellowships is to aid in the recruitment of trained health officers, directors of special medical services, and public health engineers to help fill some of the 900 vacancies in public health medical positions and 300 vacancies for public health engineers, existing in State and local health departments over the country. The Fellowships are reserved for newcomers to the public health field, and are not open to employees in State and local health departments, for whom Federal Grants-in-Aid are already available to the States.

Applicants for Fellowships may secure further details by writing to the Surgeon General, U. S. Public Health Service, Attention: Public Health Training, 19th and Constitution Avenue NW., Washington 25, D. C. Owing to the anticipated heavy enrollment in graduate schools, completed applications for training in the fall term of 1946 should be filed promptly. The awards committee will act on applications on the following dates: June 15, July 1, July 15 and August 1.

OBITUARIES

DR. ROLLIN H. STEVENS

Dr. Rollin H. Stevens died of leukemia, after a prolonged illness, on May 17, 1946. Dr. Stevens was born in 1868 in Blenheim, Ontario. He received his degree in medicine in 1889 from the University of Michigan. Early in his career he became interested in radiology and quickly became a leader in his chosen field. He was one of the first to organize a hospital x-ray department in Michigan, and was the first doctor in the middle west to use radium in the treatment of disease.

To honor his enthusiasm, his loyalty to his profession and his many accomplishments, the Journal of Radiology, on his seventieth birthday, dedicated an entire issue to him. This was known as the Rollin Howard Stevens birthday issue.

Dr. Stevens became Radiologist to Grace Hospital, Detroit, in 1903, and Consulting Radiologist to the Wayne County Hospital in 1933. He was Chairman of the Wayne County Cancer Committee in 1939 to 1940, Trustee of the American Board of Radiology in 1934, and served as Secretary of the Radiological Research Institute. He was a Diplomate of the American Board of Internal Medicine and a Fellow of the American College of Physicians since 1920.

DOUGLAS DONALD, M.D., F.A.C.P.,
Governor for Michigan

DR. ROBERT WILSON

Robert Wilson, Sr., A.B., M.D., LL.D., D.C.L., Dean Emeritus and Professor Emeritus of Medicine, Medical College of the State of South Carolina, Charleston, S. C., died of coronary occlusion and pneumonia on May 20, 1946, after a brief illness.

Dr. Wilson was born on August 23, 1867, at Stateburg, S.C. He attended the College of Charleston and the University of South Carolina (then South Carolina College), where he received the A.B. degree. After receiving the M.D. degree from the Medical College of the State of South Carolina in 1892, he entered private practice in Charleston. He became attached to the teaching staff of the Medical College in 1893 and advanced through the ranks to the professorship of medicine in 1913, at which time he played a major rôle in the transfer of the school to state ownership and operation.

He became dean in 1908, a post which he held until December 1943, when he resigned the deanship and professorship of medicine and became special lecturer in medical history. Along with his medical school positions went membership in the staff of the Roper Hospital of which he was Physician-in-Chief from 1913 to 1943.

Always active in medical organization affairs, Dr. Wilson had served as president of the South Carolina Medical Association and the Southern

Medical Association. He was First Vice-President of the American Medical Association in 1910–11. A Fellow of the American College of Physicians since 1923, he served as a member of the Board of Governors until 1936. He was a diplomate of the American Board of Internal Medicine and was a member of the American Society of Tropical Medicine, the National Association for the Study and Prevention of Tuberculosis, the American Climatological and Clinical Society, as well as the usual local and national medical associations. He also belonged to Sigma Alpha Epsilon, Phi Chi and Phi Beta Kappa fraternities.

Deeply interested in civic matters, he was Chairman of the South Carolina State Board of Health from 1907 to 1935. In 1939 he was awarded the American Legion's distinguished service plaque.

He had received the honorary degree of LL.D. from the College of Charleston and the University of South Carolina and the D.C.L. from the University of the South.

A life more complete in service and accomplishment is seldom recorded among us.

KENNETH M. LYNCH, M.D., F.A.C.P.,
Governor for South Carolina

DR. LOUIS HAMMAN

On April 28, 1946, Baltimore awakened with a shock to learn of the death of Dr. Louis Hamman. Dr. Hamman had long been associated with the College, and his interest in the work of the College in Maryland was always a great help to those in charge.

Dr. Hamman was born December 21, 1877. He obtained his A.B. degree in Rock Hill College, Ellicott City, Maryland, graduated from the Johns Hopkins University School of Medicine in 1901, did postgraduate work in Berlin, returned to the United States and became a house officer in New York Hospital, 1901–1902. For many years Dr. Hamman has been Associate Professor of Clinical Medicine at the Johns Hopkins University School of Medicine and visiting physician at the Johns Hopkins Hospital of Baltimore.

Dr. Hamman was a Fellow of the College and at one time member of the Board of Internal Medicine. He took a prominent part in the Association of the American Physicians. He was a member of the Society for Clinical Investigation and a Fellow of the American Medical Association. He published many papers and wrote the chapter on "Diseases of the Bronchi," Oxford University Press.

To those young men who have graduated from the Johns Hopkins Hospital it will be with a great deal of sorrow that they learn of Dr. Hamman's death. Baltimore and American medicine can ill afford to lose many of this type of man.

WETHERBEE FORT, M.D., F.A.C.P.,
Governor for Maryland

DR. DANIEL L. DOZZI

Dr. Daniel L. Dozzi, a Fellow of The American College of Physicians since 1941, died on May 18, 1946.

He was born on May 11, 1907, at Cardiff, Colorado. He received the A.B. degree from the University of Utah in 1929 and the M.D. degree from the University of Pennsylvania in 1932.

Dr. Dozzi did postgraduate work at the University of Pennsylvania and received the degree of M.M.S. in February 1939, and D.M.S. in June, 1939. From 1935 to 1939 he was a Clinical Assistant at Graduate Hospital. He was Instructor in Medicine in the Graduate School of Medicine at the University of Pennsylvania in 1939. From 1937 to 1939 Dr. Dozzi was Assistant Physician to the Chestnut Hill Hospital and in 1939 was appointed as Associate Physician to that hospital. Since 1939 he was affiliated with the Department of Surgical Research in the University of Pennsylvania.

Dr. Dozzi was a member of the Philadelphia County Medical Society, Medical Society of the State of Pennsylvania and the American Medical Association.

His passing at the peak of a brilliant career with a promising future is a great loss to the medical profession and the community.

EDWARD L. BORTZ, M.D., F.A.C.P.,
Governor for Eastern Pennsylvania

DR. FREDERIC MOIR HANES

In the untimely death of Dr. Frederic Moir Hanes, Professor of Medicine at Duke University, North Carolina medicine suffered the loss of one of its most distinguished members. Naturally endowed with great talents and resources he used them to the fullest extent. He had an amazing sense of values, and applied the same rigid standards of intellectual honesty, ethics and training to himself as he did to others. He knew and was personally interested in all who worked for him and with him, and the acts of kindness and consideration that he quietly extended over a period of years are almost unnumbered. I can think of few others who in appearance, intellect, training, ability and versatility could compare with him. I had a profound admiration for this truly great and versatile man whose life added so much to his distinguished family, his State, his profession, his students and the institution that he represented.

The following obituary was written for the North Carolina Medical Journal by Dr. David Cayer, of the Bowman Gray School of Medicine, who was one of his students and residents, and published by his life long friend, Dr. Wingate Johnson. Not being able to improve upon it, I submit it for publication in the official organ of the American College of Physicians of which Dr. Hanes was a distinguished member.

PAUL F. WHITAKER, M.D.,
Governor for North Carolina

On March 25, 1946 the medical profession suffered the loss of an outstanding colleague, Dr. Frederic Moir Hanes, F.A.C.P., and the North Carolina Medical Journal lost one of the most valuable members of its editorial board. Rarely does any one combine so well the necessary qualities for competence in medicine listed by Hippocrates: a natural disposition, instruction, a favorable position for the study, early tuition and love of labor.

A simple enumeration of the honors which came to Fred Hanes in his lifetime would alone testify to the position of esteem which he held among his associates. His scholastic achievements were recognized early by his election to Phi Beta Kappa. While a medical student at Johns Hopkins, he described the rare condition of familial telangiectasia with such clarity and conciseness that no one has improved upon his original work. Dr. Thayer, renowned professor of medicine at Johns Hopkins, spoke of Dr. Hanes as the most brilliant student to come out of Hopkins.

During the period from his graduation until the beginning of World War I, Dr. Hanes delved into many fields of medicine, both in this country and abroad, and laid the foundation of his unusually broad knowledge of the subject. He was for a while associated with the Rockefeller Institute, where he investigated fat metabolism and digestion, the motility of cancer cells, and bacteriologic problems, developing and utilizing technic of tissue and check embryo culture which today are among the most valuable methods in such research. When the World War began, he left the position which he then held as professor of therapeutics at the Medical College of Virginia to become commander of Base Hospital No. 65, which served with such distinction in France from 1917 to 1919. At the end of the war he returned to his native city of Winston-Salem to practice internal medicine.

In 1933 Dr. Hanes was asked to head the Department of Medicine at the Duke University School of Medicine. Although few men of his age would have attempted to return to academic medicine, and even fewer would have been able to do so, his adaptability, versatility, and remarkable ability to stay abreast of medical progress were more than adequate for the task. He fostered research projects and participated in them, contributed scientific articles to leading journals, and was the author of sections on sprue and fungus diseases in well known systems of medicine.

He did not, however, consider these endeavors as his most valuable contribution to medicine. When on occasion he was praised for his accomplishments, he was wont to point to his house staff and students and say, "These, I hope, will be my greatest contributions." He was proud of the young men whom he helped to train, and took a truly paternal interest in them. He demanded of them the same high standards of integrity and efficiency that he did of himself. Often, rather than allow a promising house officer with inadequate experience to begin the practice of medicine, he would provide sufficient funds to enable him to continue his hospital training. Most of his many kind and generous deeds were done so quietly that they will remain unknown. Many persons in need of medical and surgical atten-

tion came to the hospital bearing a note signed in his distinctive hand: "Please admit to the hospital and send bill to me."

Dr. Hanes was an outstanding example of intellectual honesty and was intolerant only of sham or pretense. In spite of a full devotion to his profession, he possessed a versatility which characterizes truly great men. He was a lover of art, a bibliophile, and an authority on Samuel Johnson. His interest in horticulture is reflected in the beauty of the Sarah Duke gardens, and in the fragrant plants which graced his offices.

Dr. Fred Hanes exemplified the highest type of clinician, teacher, and friend. His dynamic personality, his intellectual honesty, his keen and discriminating mind, and his devotion to the best in medical education all combine to make a splendid heritage for North Carolina medicine.

DAVID CAYER, M.D.

DR. CHARLES SAMUEL KIBLER

With the death of Charles Samuel Kibler, M.D., S.B., F.A.C.P., Arizona has indeed lost one of its first citizens and medical leaders for about 25 years. Dr. Kibler was born in Newark, Ohio on July 25, 1889. He did his premedical work and first year of medicine in the Ohio State University, completing his medical work and receiving his degree at Rush Medical College in 1914. Following his two years of internship at Cook County Hospital and a year of residency in the Presbyterian Hospital in Chicago, Dr. Kibler entered the medical service of the army, being discharged in 1919 as a Captain. He was Assistant Medical Chief of the Base Hospital at Camp Shelby, Mississippi while in the service.

He came to Tucson where he engaged in the active practice of medicine and continued there until his death. He practised his specialty of internal medicine, being a diplomate of the American Board of Internal Medicine. He held many offices in the Medical Societies. He was a former President of the Pima County Medical Society, former Governor for Arizona of the American College of Chest Physicians, Arizona representative on the Board of Directors of the National Tuberculosis Association, member of the American Trudeau Society, and American Association for the Study of Allergy, and Fellow of the American College of Physicians since 1926.

Dr. Kibler published numerous papers dealing with tuberculosis, allergy, and kindred subjects. Dr. Kibler was very active in the practice of medicine right up to the day of his death which was by coronary occlusion.

His wisdom and medical acumen have ever been an inspiration to his colleagues. He will be deeply missed.

FRED G. HOLMES, M.D., F.A.C.P.
Governor for Arizona

DR. ALEXANDER BERKELEY MOORE

Dr. Alexander B. Moore, F.A.C.P., one of the most widely known radiologists of America, died in Emergency Hospital, Washington, D. C., on March 8, 1946.

Dr. Moore was born in Aldie, Virginia, in 1883. He obtained his medical degree at the University of Virginia in 1907, practiced for a year at The Plains, Virginia, and for a short time in a medical clinic in Seattle, Washington. In 1909, he entered the radiological department of the Mayo Clinic with which he was identified for more than twenty years. He was a Captain in the Medical Corps of the United States Army during the First World War and at the close of the war was Chief Radiological Consultant of the Second Army, A.E.F., with headquarters at Toul, France. He returned to the Mayo Clinic in 1919 and in 1926 became head of the radiological department. In 1930 he became associated with Drs. Groover, Christie and Merritt in Washington, D. C., and for the remainder of his life was head of the radiological department of the Emergency Hospital in that city.

Dr. Moore was a Fellow of the American College of Physicians, Fellow of the American Medical Association, Member of the Medical Society of the District of Columbia, Member of the American Roentgen Ray Society and Member of the Radiological Society of North America.

"Sandy" Moore will long be remembered by his medical colleagues as a radiological diagnostician of unusual ability, especially in diseases of the chest and gastrointestinal tract. The loss of a good comrade and kindly human companion for whom they had a deep abiding affection will be felt by friends throughout the nation.

ARTHUR C. CHRISTIE, M.D., F.A.C.P.

DR. WALLER S. LEATHERS

Dr. Waller S. Leathers, F.A.C.P., died in Nashville, Tennessee, January 26, 1946. He was born in Virginia in 1874. He graduated in Medicine from the University of Virginia in 1895 and took postgraduate work at Johns Hopkins, The University of Chicago and Harvard Medical School.

After serving as Professor of Chemistry at the Miller School of Virginia 1896–1897 Dr. Leathers became Professor of Biology at the University of South Carolina. In 1899 he accepted the chair of Zoology at the University of Mississippi and later was made head of the Department of Physiology and Hygiene. His work became so outstanding that he was then made Dean of the Medical Department.

From 1910–1924 Dr. Leathers served as Director of Public Health in the State of Mississippi. It was during this period that Mississippi made its greatest advances in public health.

In 1924 he was called to the faculty of Vanderbilt University School of Medicine, becoming its Dean in 1928.

In 1918 he was president of the Mississippi State Medical Association.

In 1922 he became president of the Southern Medical Association. It was in 1923 that he was made a Fellow in the American College of Physicians. He was President of the Tennessee Academy of Science in 1926; vice-president of the American Association for the Advancement of Science in 1928; President of the American Public Health Association in 1940 and President of the Association of American Medical Colleges in 1943.

Under the skilled leadership of Dr. Leathers the Vanderbilt Medical School became one of the outstanding medical schools of the country. His great interest in preventive medicine and public health and his untiring work in those problems pertaining to sanitation and health were responsible for great improvement in living conditions in the entire South.

The many honors that came to Dr. Leathers were in recognition of his leadership in public health, in medical science and in better medical education.

WILLIAM C. CHANEY, M.D., F.A.C.P.,
Governor for Tennessee

DR. EUGENE ROSAMOND

Dr. Eugene Rosamond, F.A.C.P., died in Memphis, Tennessee, Dec. 12, 1945. He was accidentally shot in the cervical region of his spine in 1940, resulting in paralysis in all four extremities.

Dr. Rosamond was born at Cedar Chapel in 1880. He received the degree of A.B. at Ouachita College, Arkansas, in 1899, and then studied Medicine at the University of Louisville, Kentucky, where he graduated in 1905. In preparation for specializing in Pediatrics Dr. Rosamond did postgraduate work at the New York Polyclinic Hospital and New York Postgraduate Hospital.

He was a member of the Central States Pediatric Society, The Memphis and Shelby County Medical Society and the American Medical Association. He became a Fellow of the American College of Physicians in 1929.

For many years Dr. Rosamond had been one of the leading Pediatricians in the South. His comprehensive knowledge of his specialty together with an ability to talk well were responsible for his appearing frequently upon medical programs all over the country.

WILLIAM C. CHANEY, M.D., F.A.C.P.,
Governor for Tennessee

DR. DONALD BLAIR LOWE

Donald Blair Lowe, A.B., M.D., F.A.C.P., died in Akron, Ohio, March 2, 1946, of acute hemorrhagic pancreatitis.

Dr. Lowe was born August 1, 1883, at Kingsville, Ohio. He attended Western Reserve University in Cleveland where he received his A.B. degree in 1909 and his M.D. degree in 1912. The following two years he served as instructor in medicine at that University. In 1915 he was appointed Director of the Medical Department of B. F. Goodrich, Akron, Ohio, which position he held during his whole life. Under his direction this Medical

Department assumed greater and greater importance and Dr. Lowe was among the foremost of the industrial physicians in this County.

He served as President of the American Association of Industrial Physicians in 1932 to 1933. His keenness of thought prompted him to suggest many industrial research problems to other investigators.

He was admitted to the Summit County Medical Society February 1, 1916, and became its President in 1939 after serving faithfully on many of its standing committees.

For many years he was a member of the staff in Internal Medicine of The City Hospital of Akron and served as Hospital Chief of Staff in 1936. He was a Fellow of the American College of Physicians and a member of Delta Upsilon Fraternity.

Dr. Lowe was one of the most highly respected physicians of this State. His activities ranged from an intense desire to teach younger doctors to participation in all matters of civic interest. He was steadfast in his opposition to sham and pretense and demanded of his associates and colleagues the same intensity of purpose which he had.

Since his death there has been set up a fund to be known as The Donald Blair Lowe Fund for educational purposes.

ALEXANDER PIERCE ORMOND, M.D., F.A.C.P.

DR. CLARENCE L. HYDE

In the death of Dr. Clarence L. Hyde, F.A.C.P., on December 1, 1945, the medical profession lost a distinguished leader known both nationally and internationally for his work in the prevention, control, and treatment of tuberculosis.

Dr. Hyde was born May 23, 1878, the son of the Reverend Melancthon Cleveland Hyde, then Rector of All Saints Episcopal Church, Buffalo, New York, and Elizabeth Ludlam (Stoutenburgh) Hyde.

He received his medical training at the University of Michigan, from which he was graduated in 1906, and later served as house officer at the Homeopathic Hospital of Montreal, Canada. In 1909, he married Baca Luz Chisholm of Montreal, who survives him, together with their two children, Cleveland Chisholm Hyde and Elizabeth Margaret Hyde.

From 1909 to 1911, Dr. Hyde served as Director of the Division of Tuberculosis in the Dept. of Health of Buffalo, New York. During his residence in Buffalo, he was also Consultant in Tuberculosis at the Ernest Wende Hospital and at Buffalo City Hospital. His unusual ability was quickly appreciated and he was called to be Superintendent of the J. M. Adam Memorial Hospital, Perrysburg, New York, where his work from 1913 to 1920 received national attention. In the latter year, Dr. Hyde became Superintendent of the Edwin Shaw Sanatorium, Akron, Ohio, where he remained until the time of his death.

Dr. Hyde was one of the initiators of the county-wide plan for the control of tuberculosis. Soon after taking up his duties in Akron and Summit

County, he established the Municipal Tuberculosis Clinic with subsidiary clinics in the smaller communities where from the very beginning a chest roentgenogram was a part of every examination. He was an early advocate of the use of collapse therapy measures and at the same time stressed the value of absolute and prolonged bed rest. He was recognized widely as a pioneer in the use of heliotherapy in America for the treatment of extrapulmonary forms of tuberculosis. He attacked the problem of tuberculosis control by means of continuous and effective education of the public, a meticulous case-finding program, and a modern and attractive tuberculosis hospital where treatment and rehabilitation of the patient were carried out scientifically and sympathetically.

The soundness of Dr. Hyde's ideas is amply attested by the results of his labors. The electorate of Akron and Summit County has repeatedly approved tax levies to support the Sanatorium by majorities as great as 85 per cent; the per capita seal sale has increased each year to become the highest of all the metropolitan counties in Ohio; the percentage of cases of tuberculosis diagnosed in the incipient stage of the disease has risen steadily; finally, the tuberculosis death rate in Akron has decreased until it is one of the lowest, if not the lowest, among the 42 large cities of the nation.

Dr. Hyde was a fellow of the American College of Physicians, the American Medical Association, the National Tuberculosis Association, and the American Trudeau Society. He served as President of the Summit County Medical Society in 1928. In 1934, he was an official delegate representing the United States to the Ninth Conference of the International Union Against Tuberculosis held in Warsaw, Poland, where he discussed "Treatment of Tuberculosis of the Bones and Joints."

Dr. Hyde will be remembered not only for his distinguished medical career but also for the imprint of his life and character upon an amazingly wide circle of friends. A genial, kindly man with an extraordinary capacity for friendship, Dr. Hyde radiated confidence. He helped many through critical times by the faith he inspired in them. All through his life, his active leadership in the church reflected his early training in a clergyman's home. He was the embodiment of Christian kindness and courtesy and one of the most beloved men in the communities he served so well.

The Rector of St. Paul's Parish, Akron, in which Dr. Hyde served as Vestryman for over twenty years, says of him:

"He was easily the St. John of our Vestry, indeed the most spiritually minded man I have ever known. His goodness was never heavy. People in all walks of life loved to be near him, and found themselves enriched by his friendship. He was eternally young in spirit. His hearty laugh was like a tonic. As I think of him I recall a line of poetry which at the moment I cannot identify—'No beggar ever felt him condescend, nor prince presume.' He was humble, as all great men are, and like the Master he served so well, 'went about doing good.' May light perpetual shine upon him!"

THEODORE L. BLISS, M.D., F.A.C.P.,
Akron, Ohio